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The Journal of Thoracic Surgery

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The Journal of Thoracic Surgery

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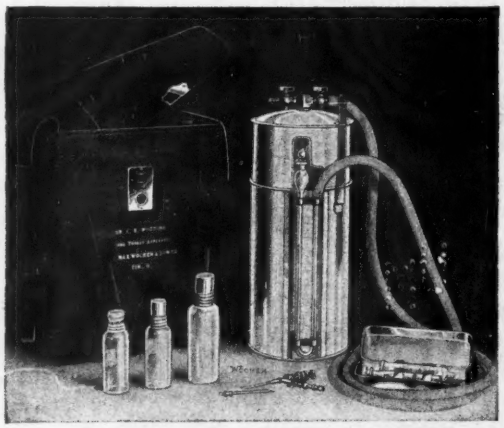

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The Journal of Thoracic Surgery

VOL. I OCTOBER, 1931 No. 1

Foreword

THE field of thoracic surgery has been developed enormously since the Great War. Realizing the fascinations and potentialities of surgical work in the chest, Dr. Willy Meyer of New York in 1917 called together a group of men to discuss the formation of a special organization for the study and discussion of problems related to this field. It was fitting that the impetus should come from Dr. Meyer; for in America he has been a Nestor to all those interested in this work. As a result of this meeting the American Association for Thoracic Surgery was formed. The annual meetings of this Association during the past fourteen years have constituted the chief forum on this continent for the discussion of problems and experiences in this special field by those who have been most interested in making surgical interventions in the thorax not only safe but feasible as well. The discussions at the meetings, however, have not been confined to matters which might be considered as purely surgical, but in addition much new light has been thrown on many of the fundamental problems of physiology, bacteriology and pathology which are concerned. Contributions of immense significance to this Association have been made by the splendid group of younger, scientifically trained surgeons who have profited by the opportunities afforded by the revolution in the medical schools in this country to obtain a proper groundwork before entering upon surgical careers. Many of these contributions have been the result of important and painstaking experimental research. It is noteworthy also that many members of the Association are not surgeons at all but are physicians, roentgenologists, bronchoscopists, etc., interested in the diverse problems of thoracic disease.

Soon after the formation of the Association it became apparent that it would be desirable to publish its papers and transactions together in some suitable journal. Accordingly after the 1922 meeting arrangements were concluded with the Archives of Surgery to publish each year a special supplement devoted to the affairs of the Association. This arrangement has been of the utmost help in obtaining a suitable

audience for the papers presented at the meetings. Nevertheless there has been a growing desire among the members to have a journal in the English language devoted to the special field of thoracic surgery which might also be the official organ of the American Association for Thoracic Surgery. This new journal has sprung into being to fill this apparent need. With our first issue we wish to pay our respects to the only other journal dedicated to this field, *Archives Médico-Chirurgicales de l'Appareil Respiratoire,* the first volume of which appeared in 1926.

It is the desire of the editorial board that THE JOURNAL OF THORACIC SURGERY shall attract to it suitable articles bearing on all phases of thoracic disease. Its pages will not be restricted to those papers which have been presented at the meetings of the American Association for Thoracic Surgery. Especially welcome will be articles dealing not only with the purely clinical aspects of this field but also with reports of carefully conducted experimental investigations in the realms of physiology, bacteriology, pathology, etc., which may have a bearing on the clinical problems concerned.

The new journal will appear bimonthly and will contain about 600 pages in the volume. In addition to the original articles there will be selected abstracts, book reviews, and a few pages devoted to news of the American Association for Thoracic Surgery.

—Editor.

Original Communications

POSTOPERATIVE ATELECTASIS AND COLLATERAL RESPIRATION*

C. M. VAN ALLEN, M.D., AND T. S. JUNG, M.D.

AT THE last meeting of the Association, Van Allen and Lindskog[1] described briefly a newly recognized property of the lungs. They showed that the lobular divisions of the pulmonary air passages in dogs are connected collaterally, rather than being independent as generally supposed. The interlobular septa are incomplete and the alveoli at the planes of fusion of the lobules communicate with each other by minute openings (probably the alveolar pores of Kohn). The communication comes spontaneously into use when the bronchi supplying one lobule or group of lobules becomes obstructed, for then the obstructed part breathes from the side through the adjacent free parts of the lobe. No such connection as this exists between the lobes, for the interlobar septa and fissures completely separate them. Therefore, when the bronchus of an entire lobe is obstructed, the air passages are entirely isolated.

During the past year, these authors and their associates, Richter and Ch'in,[2, 3, 4, 5, 6] studied this property of the lungs more completely, and they termed the function arising therefrom "collateral respiration." They demonstrated in dogs that the interlobular connection is exceedingly abundant and suffices for maintenance of inflation and appreciable function in an obstructed segment of lung lobe. Lobular bronchial obstruction was found not to produce atelectasis, whereas lobar bronchial obstruction did so. Also, proof was secured in the lungs of man of the presence of interlobular connections and of the occurrence of collateral respiration.

Collateral respiration appears to have much the same economic significance in pulmonary function as does collateral circulation in the function of the vascular system. The bronchioles are fine and possess richly secreting surfaces. Lobular bronchial obstruction probably occurs to some degree with every attack of severe lower respiratory catarrh. While the individual is conscious and able to respond with vigorous breathing and coughing, the airways are likely to be kept well cleared; but during periods of sleep with breathing shallow and

*From the Departments of Surgery and Radiology, Peiping Union Medical College, Peiping, China.

Read at the Fourteenth Annual Meeting of the American Association for Thoracic Surgery at San Francisco, July 1, 2, 3, 1931.

coughing quiet for hours, the secretions must tend to accumulate, gravitate and fill some of the slender ducts at the base of the lungs with columns of fluid, blocking the passage of air. After bronchial obstruction has occurred the imprisoned air is known to undergo absorption in an hour or so to such extent that, unless air has been constantly supplied by collateral respiration, so little air remains in the occluded part of the lung that cough is rendered ineffectual in expelling the obstruction.[4] Therefore, were it not for collateral respiration—that is, were the pulmonary lobules possessed of blindly terminating passages—the operation of the lungs in the presence of bronchitis would be inefficient. It would be comparable to the operation of a blood-vascular system without collateral circulation.

Since lobular atelectasis may occur from spontaneous bronchial obstruction, in both man and animals, it is evident that collateral respiration is occasionally prevented. The work to be presented in this paper was carried out to determine and evaluate the factors which may interfere with collateral respiration. Some factors of possible significance in this respect occurred to us from analysis of the physical circumstances of that class of individuals which most frequently develops lobular atelectasis of sufficiently massive proportions for clinical diagnosis; i.e., patients recovering from major surgical operations. First, attention is called to the fact that these patients breathe lightly. William Pasteur[7] observed this in 1914. In uncomplicated cases convalescing from appendectomy, he found that the diaphragm was relaxed and moved very little, even with deep breaths. Costal excursions were less limited. The observations were verified at that time by Hardy.[8] Through the more recent work of Harries,[9] Featherstone,[10] Muller, Overholt and Pendergrass,[11] Patey,[12] and Jung and Van Allen[13] this type of diaphragmatic paresis is known to occur with special frequency during convalescence from laparotomy, particularly when the incision involves the upper abdomen. Deficient respiratory depth is evidenced in these patients by marked reduction of vital capacity, as shown by Churchill and McNeill,[14] Head,[15] Powers,[16] Patey,[12] and Overholt.[17] The cause of the specific depression in diaphragmatic function and vital capacity is beyond the scope of the present discussion. The second circumstance to be emphasized is that those patients who develop atelectasis after operation have excessive bronchial exudation before, during, and after the disease. Quantities of thick, sticky sputum appear. The third, is that those who develop atelectasis give evidences of deeply implanted pulmonary infection, always with fever and often with frank bronchopneumonia. A direct causal relationship between these factors and atelectasis is now commonly accepted, on the ground that poor diaphragmatic function results in deficient aeration of the base of the lungs and weakened cough, and that these lead to retention of inflammatory secretions and to lobular bronchial obstruction. We be-

lieved this to be so far correct, but since we knew that lobular bronchial obstruction *per se* did not lead to atelectasis, we felt that interference with collateral respiration was a necessary step in the process. It seemed probable that the same special physical circumstances which lead to blockage of direct respiration of the lobules may, in exaggerated form, interrupt collateral respiration. That is, it seemed probable that at times the secretions in the bronchial tree may extend to the fine airways at the interlobular boundaries, shutting off collateral exchange of air; that the parenchyma at the interlobular boundaries may become swollen and consolidated, producing the same effect; and that restricted breathing may lower the rate and volume of collateral exchange to a degree that absorption of air in the obstructed parts is not compensated.

Accordingly, the effects of these factors were tested experimentally. Lobular bronchial obstruction was produced permanently in a series of

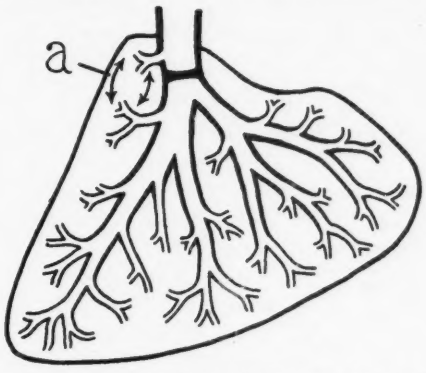

Fig. 1.—Diagram of the bronchial tree of the right lower lobe of a dog's lung, showing the site of ligation used in the experiments. Arrows, at *a*, indicate the path of collateral respiration between the obstructed and free portions.

dogs. About one-half of the animals were then treated further, some by introducing excessive amounts of fluid into the bronchi of the unobstructed parts of the lobe, others by inoculating these bronchi with a chemical or bacterial irritant, and the rest by interfering with the movements of respiration in different ways. The remaining dogs were used as controls and given no supplementary treatment. By chance, a few of the control group developed bronchitis and pneumonia, and the opportunity was afforded of witnessing the effects of spontaneously arising intrabronchial fluids and parenchymatous consolidation after lobular bronchial obstruction.

The lobular bronchial obstruction was produced in each case by ligation. The chest was opened under positive pressure breathing and with sterile technic, and the main bronchus of the right lower lobe was exposed and tightly ligated at a point just peripheral to the origin of

the first branch of the lobe. The lungs were then fully inflated and the chest was closed. Fig. 1 shows the position of the ligature. About nine-tenths of the lobe was occluded. The operation was well tolerated, except in a few cases where the ligature cut through the bronchus after four or five days and infection developed. These dogs were discarded. Otherwise, those allowed to live long enough showed the bronchus to be closed at the ligated site.

EXPERIMENTAL RESULTS

I

Twenty dogs had lobular bronchial obstruction without supplementary treatment. X-ray examination at frequent intervals showed that the lungs remained air-containing in 15 cases. Here, sacrifice and autopsy

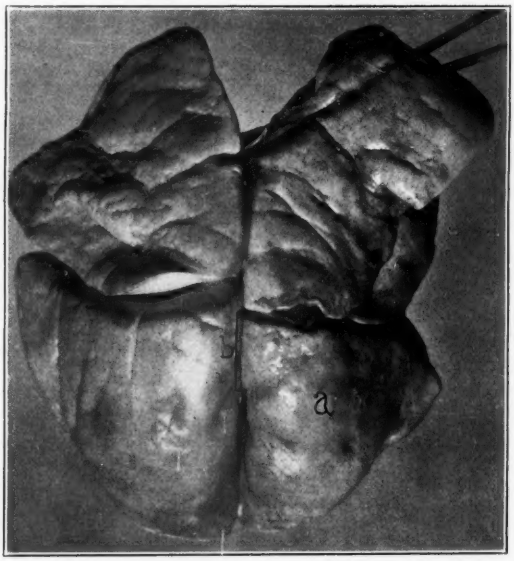

Fig. 2.—Photograph of a dog's lungs obtained three months after lobular bronchial ligation (see Fig. 1). The right lower lobe, at *a*, is completely air-containing. The free lobule is at *b*.

were performed after periods of twenty-four hours to three months, and the lungs were found to be normally aerated throughout (Figs. 2 and 3). Section of the ligated portion of the right lower lobe in each animal demonstrated that the bronchus was closed at the point of ligation and was filled with glassy mucus beyond it, and that the parenchyma was everywhere air-containing and indistinguishable grossly from that of the other lobes. Five dogs developed epidemic respiratory infection (distemper) several weeks after operation and were sacrificed during the later stages of this disease. At autopsy, quantities of thick

mucopurulent exudate were found scattered throughout the bronchial tree, including the bronchi of the free section of the right lower lobe; and there were patches of pneumonia involving various parts of the lungs. In 3 cases, the right lower lobe was normally aerated throughout and without changes except for occlusion of the bronchus at the point of ligation, and moderate congestion. The fourth had a patch of pneumonia in the free lobule of that lobe, consolidating about two-thirds of it. The larger portion of the ligated division was atelectatic

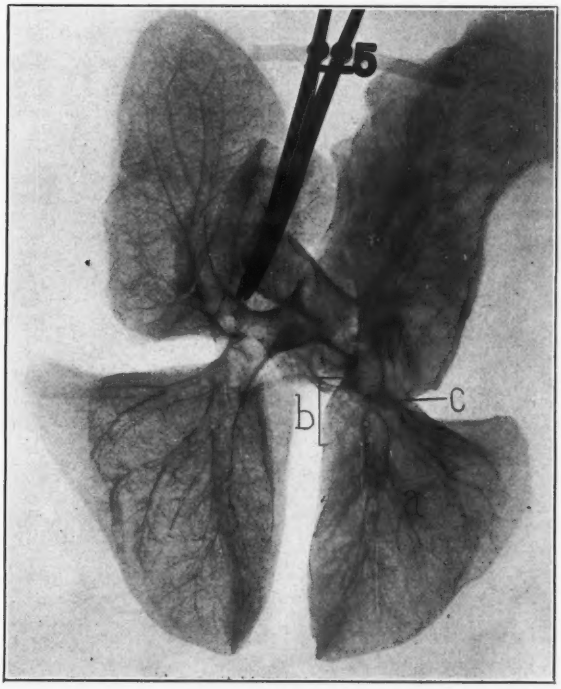

Fig. 3.—Roentgenogram of the lungs obtained two months after lobular bronchial ligation (see Fig. 1). The right lower lobe, at *a*, is completely air-containing. The free lobule is at *b*, and the point of ligation at *c*.

and the remainder was aerated. The fifth dog showed intense swelling and consolidation of the entire free lobule of the right lower lobe. The ligated division was completely collapsed and atelectatic.

These experiments indicate that collateral respiration usually maintains the inflation of obstructed sections of lung lobes, even when the sections are very large and when adverse circumstances exist, namely, thick secretions in the bronchi, areas of pneumonia in the unobstructed lobes and general pulmonary congestion. However, when pneumonia develops in the free portions of the obstructed lobes, collateral respira-

tion may be interfered with so much as to allow atelectasis to develop in the obstructed portions.

II

Thirteen dogs had lobular bronchial obstruction, followed by supplementary measures. They were sacrificed and autopsied twenty-four to seventy-two hours after the latter. Four groups were distinguished.

One group, five dogs, received right single or bilateral phrenicotomy immediately after the ligation or two to three weeks later. X-ray examination after this showed the diaphragm on the right side, or on both sides, to be elevated and fixed save for slight paradoxical movements. Where both sides were paralyzed moderate dyspnea occurred. Mor-

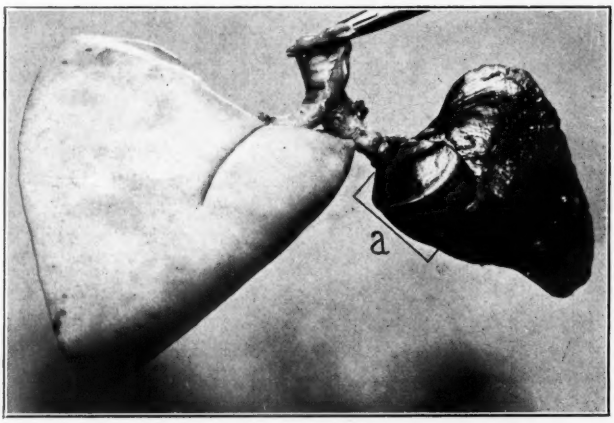

Fig. 4.—Photograph of the lower lung lobes (normally about the same size) obtained forty-eight hours after lobular bronchial ligation (see Fig. 1) and inoculation of the free lobule with pneumococcus. The free lobule, at *a*, is swollen and consolidated, and the remaining, ligated portion of the lobe is shrunken and atelectatic. The other lobe is normally inflated and without gross lesions.

phine was given frequently in large doses to depress the costal breathing. The result in every case was that the ligated division of the lobe remained fully aerated.

The second group, four dogs, was bronchoscoped immediately after the ligation and 20 c.c. of freshly drawn, autogenous blood was injected into the orifice of the free bronchial branch of the right lower lobe. The blood filled the branch and flowed backward, filling also the primary bronchus. The dogs were kept still (under anesthesia) for two hours to allow the blood to infiltrate. One dog was then roentgenographed and showed a mottled shadow in the lung at the site of the injected lobule. Daily x-ray examination and autopsy after three days proved the ligated division of the lobe to remain air-containing in all cases. The injected lobule was red and infiltrated with

blood, its parenchyma everywhere partially air-containing, and its larger bronchi patent.

The third group, two dogs, was bronchoscoped immediately after the ligation and 1 c.c. of fluid ether was injected into the free bronchus of the right lower lobe. Then the dogs were allowed to recover consciousness. One was sacrificed and examined at twenty-four hours. The whole injected lobule was found to be greatly swollen and massively consolidated with blood. The ligated division of the lobe was shrunken and the parenchyma was completely airless. The other dog was sacrificed at seventy-two hours and displayed hemorrhagic consolidation of one-half of the free section of the right lower lobe. The remainder of this part and all of the ligated division were air-containing and normal in appearance except for slight congestion.

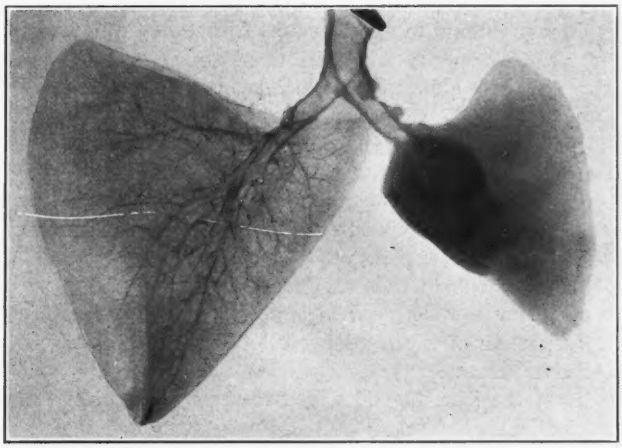

Fig. 5.—Roentgenogram of the specimen shown in Fig. 4. The pneumonic part is seen to be swollen and very dense and the atelectatic part collapsed, thin and airless, in contrast to the normal qualities of the other lobe.

The fourth group, two dogs, was bronchoscoped right after ligation and 2 c.c. of concentrated, twenty-four-hour broth culture of Type 1 pneumococcus was injected into the free bronchus of the right lower lobe. X-ray examination at twenty-four hours revealed partial or complete consolidation of the lobe and displacement of the heart and diaphragm toward it in both cases. Sacrifice and autopsy at forty-eight hours showed the free lobule of the right lower lobe to be the seat of pneumonia. In one dog, the consolidation was complete and the lobule was greatly swollen; and here the ligated division was shrunken and airless (Figs. 4 and 5). In the other, the free lobule was only partly consolidated, and the ligated portion was about half atelectatic and half aerated. The pneumonic and atelectatic regions were adjoining, and the same was true for the air-containing portions of the two di-

visions of the lobe. Test was made of patency of the collateral connections between the air-containing portions by blowing gently into the bronchus. Air passed readily from the free to the ligated parts.

The experiments of these two groups demonstrated that collateral respiration may maintain the inflation of an obstructed section of lung lobe when diaphragmatic function has been eliminated and no other form of interference is added; also, when large quantities of viscid, coagulable fluid have been deposited in the airways of the remaining parts of the lobe, without other interference. They show, however, that when the portion of the lobe without bronchial obstruction is inflamed, swollen and consolidated from chemical or bacterial action, collateral respiration may be interfered with so much as to allow atelectasis to develop in the obstructed portion.

III

Four dogs were used in experiments of somewhat different character. First of all, tracheotomy was performed and a slender rubber tube, tipped with a short glass cannula, was introduced into the trachea and passed down to the bronchus of the right lower lobe. The chest was then opened under positive pressure breathing and a ligature was passed around the bronchus of that lobe just distal to the first branch (the same point as used previously). The tip of the tube was maneuvered to this spot and was secured tightly in the bronchus by tying the ligature. The lungs were expanded and the chest was closed. The outer end of the rubber tube was fitted with a long glass nozzle, and the tip of this was submerged in water in a nearby vessel (Fig. 6). The major portion of the right lower lobe was thus obstructed with a delicate valve permitting expiration, but not inspiration, of air. The other part of the lobe, together with the remainder of the lungs, was free to breathe as before. Ether anesthesia was maintained throughout the experiment. From the beginning of the obstruction it was noted that a stream of bubbles escaped from the nozzle at each expiration. Within one-half hour a quantity of air had been expelled which was vastly greater than the capacity of the ligated division of the lobe, indicating that collateral respiration was operating to supply air to the division. Fluoroscopic examination showed the lobe to be normally inflated. The escape of air from the nozzle was then used as an index of the occurrence and rate of collateral respiration during a series of manipulations to interfere with respiratory movements, as follows:

The anesthesia was deepened and the respirations were caused to become very shallow. The amount of air which escaped from the nozzle with each breath (the rate of collateral respiration of the obstructed division) became greatly reduced. One or two bubbles escaped at a time. The anesthesia was then lightened, bringing the animal into the excitement stage of narcosis and causing it to breathe heavily. The

rate of collateral respiration was thereby much increased. Anesthesia was then regulated again to medium depth. Collateral respiration returned to the original rate.

A bandage was applied firmly to the chest (Fig. 6, *A*). This had no effect on the rate of collateral respiration except in one animal, in which the rate was definitely increased. Fluoroscopic examination showed the ribs to be compressed to a position of complete expiration and to be motionless, and the diaphragm to be moving to an exaggerated degree. The bandage was then removed and applied to the abdomen with a thick pad in the epigastrium (Fig. 6, *B*). The effect of this

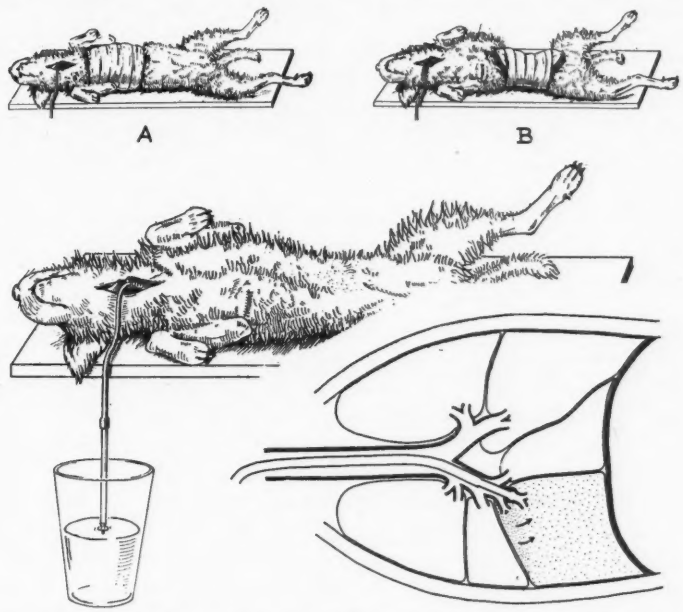

Fig. 6.—Sketch of the arrangements for the acute experiments on limitation of respiratory movements. Below: arrangements for normal breathing, showing the emergence of the tube from the trachea and its submersion in water to form a valvular type of obstruction; also (insert), a diagram of the lungs in the chest, giving the path of the tube and the site of its fixation in a bronchus of the right lower lobe. Lightly shaded portion of the lobe is the obstructed division; densely shaded portion is the free lobule. Above: at *A*, bandage applied to the chest to stop costal breathing, and, at *B*, bandage applied to the abdomen to stop diaphragmatic breathing.

was immediately to reduce the rate of collateral respiration. One bubble escaped from the nozzle every two or three breaths. Fluoroscopic examination showed the diaphragm to be elevated and very much limited, the ribs to be spread, and the costal movements to be exaggerated. The bandage was removed, and following a few deep abdominal breaths collateral respiration returned to about its initial rate.

Both phrenic nerves were then exposed in the neck and avulsed. The escape of air at the nozzle was immediately reduced to one bubble

every two or four breaths. Fluoroscopic examination demonstrated elevation and flacidity of the diaphragm and increased costal excursions.

One dog prepared for use in this type of experiment reacted differently. The bubbling from the nozzle was much less voluminous from the start than with the others. Limiting the movements of the chest wall did not affect it appreciably, but constriction of the abdomen completely eliminated it. The chest was reopened to determine the cause of the reduced efficiency of collateral respiration. The right lower lobe was congested and edematous and presented many petechial hemorrhages, particularly in the region of the free lobule. It had apparently been traumatized more than usual at the operation.

These experiments made it clearer that the rate of collateral respiration of an obstructed part of the lower lobe is controlled by the movements of respiration, much more by those of the diaphragm than by those of the ribs. Total loss of diaphragmatic movement reduces the rate of this function markedly but not entirely, that is, when no other pathological changes are present; but when the lobe is congested and edematous and collateral respiration is already reduced in efficiency, limitation of diaphragmatic excursions may stop the function.

DISCUSSION

Collateral respiration was found to be surprisingly efficient. A very small section of lung lobe proved to be sufficient for aeration of the whole. Continuation of aeration of the ligated division when the bronchi of the free section were flooded with viscid fluid (blood) can be explained only by supposing that the fluid was soon absorbed or disseminated to a large degree. Nevertheless, judging from the diffuse red color and distinct swelling of the injected lobule, it was evident that much of the fluid still remained in the peripheral airways. The normal capacity of even this small section of the lobe seems, therefore, to have been superabundant for maintaining the inflation. Also, a wide margin of safety was present as regards the amplitude of respiratory movements, since very shallow breathing afforded significant collateral exchange. The most effective obstacle to the function was inflammatory consolidation of the tissues at the border between the obstructed and free sections of the lobe, although here, too, there was remarkable tolerance. The tissues had to be practically completely consolidated before this factor *per se* eliminated interlobular exchange altogether. Concluding from the last experiment cited, it is likely that combinations of the three factors might eliminate collateral respiration when one of them acting alone would not do so.

The greater dependence of collateral respiration in the lower lobe upon movements of the diaphragm than upon those of the ribs is well explained by what is known of the mechanics of breathing. As Keith[18] pointed out, "The upper set of ribs is . . . raised by a bucket-handle

movement, thus expanding the chest laterally. The lower set undergoes no such movement; they are long levers articulated at their vertebral ends. When their ends are raised, they lead to a great lateral enlargement of the abdominal wall but only to a very partial enlargement of the real pulmonary space. That is to say, while the upper set are direct lateral distenders of the lung, the lower are made accessory to the diaphragm. They provide a fulcrum for the diaphragm; and their lateral movement is designed to make room for the abdominal viscera displaced by this structure, rather than to cause a direct enlargement of the lung.'' Accordingly, the descent of the diaphragm is almost entirely responsible for the expansion of the lower lobes, and it is apparent why loss of this motion is more deleterious to the efficiency of collateral respiratory exchange in those parts of the lungs than loss of costal motion. Since the ribs are the principal expanders of the upper lobes, it is likely that their immobilization would be more harmful to collateral respiration in the upper lobes than would immobilization of the diaphragm. This point was not investigated, because in postoperative atelectasis we have to do chiefly with involvement of the lower lobes and with diaphragmatic paresis.

Swelling of the interalveolar septa is known to occur to a marked extent in pneumonia, even before filling of the alveoli with exudate develops.[19] Since collateral respiration depends upon the patency of minute channels through these septa, one reason seems furnished for the special tendency of parenchymatous inflammation to interfere with this function. Another reason, of course, is consolidation of the alveoli.

We have come to the conclusion that interference with collateral respiration constitutes a necessary link in the sequence of events in the development of most instances of postoperative atelectasis. It is very likely that every patient who develops severe bronchorrhea acquires bronchiolar obstruction of some degree and of significant duration, but atelectasis forms only when collateral respiration is interfered with, as may well happen under the special circumstances attending convalescence from abdominal operations.

SUMMARY

The pulmonary function of collateral respiration is described. Also, the sequence of events in the pathogenesis of postoperative atelectasis is explained, as now generally understood. It is demonstrated by presentation of new experimental evidence why consideration of collateral respiration and factors which reduce its efficiency must be included in the pathogenesis. These factors are named and evaluated.

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DISCUSSION

DR. W. E. LEE.—As usual, Dr. Van Allen's contributions leave little for one to discuss, but provide facts for which many of us have been searching. Dr. Van Allen brought this matter of the collateral alveolar circulation to the Philadelphia Meeting of the American Surgical Association last year, and it seemed to us at that time to supply the missing link in the pathogenesis of obstructive pulmonary atelectasis which has made many doubt our explanation of its etiology.

The fact that occasionally, in some of our experimental work we failed to produce massive atelectasis by introducing viscid substances into the bronchial tree can certainly be explained by this phenomenon of collateral alveolar circulation.

Step by step we feel that the etiology of the phenomenon of obstructive pulmonary atelectasis is being developed, and though even at the present time we may not have the complete story, the knowledge that we do possess of the etiologic factors involved certainly removes the dread of postoperative pneumonia from the surgeon, and in our own personal experience has practically eliminated it as a postoperative complication.

DR. S. BRILL.—I think this work helps explain the experiments which we demonstrated in the laboratory yesterday. During the past year or so, we have developed a method by which we can produce mucous plugs in the bronchi of dogs by irritation. We were surprised to find that even when the bronchi were plugged atelectasis did not occur except in one instance. In this experiment there occurred a thrombosis of the vessels and an infarction on which was superimposed infection. These specimens were demonstrated yesterday. I now believe that this paper explains why atelectasis did not occur in our other experiments.

DR. E. HOLMAN.—May I say a word in appreciation of what Dr. Van Allen is doing in Peiping? He is developing a department of thoracic surgery in an almost totally virgin field, and he has a great deal of material upon which to work. Furthermore he has unlimited facilities in the laboratory, and one may expect even more important contributions from his facile pen and alert mind. This study of collateral respiration seems to be a contribution as important to thoracic respiration as was John Hunter's memorable observation on the collateral circulation in the horns of the deer.

THE MORTALITY OF OPERATION FOR ACUTE EMPYEMA*

GEORGE P. MULLER, M.D., PHILADELPHIA

THE necessity for the evacuation of pus in the chest has been recognized since the time of Hippocrates and through the centuries that followed there has been constant repetition in the advocacy of different methods of procedure. Until the last hundred years the diagnosis of empyema was largely a matter of guesswork so that the high mortality, said to be 90 per cent, was dependent upon the sepsis and prolonged suppuration from chronicity and perhaps the result of the wide intercostal incision made with the cautery.

In 1873 Playfair advised aspiration, and the method was developed by Dieulafoy and Potain, but the former could not have been much impressed by its success because he chose to die, unoperated upon, from empyema. Hewitt (1876) suggested continuous aspiration. In recent years aspiration has been used mostly as a preliminary step to drainage, although McEnery and Brennemann (1929) have had a remarkable series in infants with a mortality of 6 per cent only. In 1875 various writers advocated aspiration and irrigation with solutions of carbolic acid or iodine, and comparatively recently formalin and glycerine, methyl violet, ethylhydrocupreine, etc., have been used.

In 1878 Roser advised resection of a rib for better drainage if there was any difficulty in clearing the chest of pus through the aspirating trocar, and Konig (1878) advocated rib resection as a routine procedure. This became the routine method of treatment and by 1900 the reported mortality was around 30 per cent. At the Eighth German Congress for Medicine in 1903 aspiration and irrigation were rejected and the argument lay between Bulau's (1891) method of interrib puncture with siphon drainage and rib resection. In 1898 Perthes added a suction pump to the drainage mechanism. In 1911 Tigel devised a metal drain with valves, and others advocated various kinds of rubber valved tubes. At this Congress it was agreed that any method must (1) remove the pus, (2) prevent its regathering and, (3) restore the respiratory apparatus in its anatomical and physiological relations as completely as possible. It is interesting to recall that Aufrecht reported that the lung will expand if the diameter of the fistula or opening in the chest wall is smaller than that of the bronchus. This and some other experimental work reported at that Congress were basic but

*From the Surgical Division (Service B) Hospital of the University of Pennsylvania.

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no one appreciated their importance until the work of Graham (1918) in later years.

As early as 1882 Parker suggested pneumothorax as a treatment for empyema and eight years later Forlanini (1890) advocated aspiration of the pus and the introduction of air to be followed later by interrib suction drainage. This method was taken up by Morelli in 1918 and his method was explained by Davis (1920) a few years later. Recently Danna (1931) has reported a series of 35 cases treated by this method.

In 1915 Wilensky reported the results of drainage for acute empyema at the Mt. Sinai Hospital, New York, during the years 1903-1913 with a mortality of 28 per cent and this may be taken as the average figure for that time. They practiced rib resection, used two tubes for drainage, did not practice irrigation and noted no encouragement from suction drainage. Wilensky makes the significant statement that the operative mortality was highest when the pus was thin and had been present only for twenty-four or forty-eight hours.

Then came the war, the epidemics, the great number of empyemata, the report of the empyema commission and the classic experiments of Graham and Bell (1918). Two statements from the report of the commission need emphasis: First, the evacuation of the pleural exudate by operation early in the disease involves greater risks, without compensating benefits, than the removal by aspiration, and secondly, the drainage of the empyemic cavity after operation should be both complete and continuous. They advised local anesthesia and irrigation with Dakin's solution in the absence of bronchial fistula. Numerous reports then appeared detailing the experience of the various base hospitals, and Mozingo attracted attention by his rediscovery of aspiration and irrigation.

Naturally, the experience of the army camps had an echo in civilian practice and during the next decade numerous reports appeared showing a lowering of mortality, but curiously all writers discussed open versus closed drainage when nobody left tubes opened up, and classed rib resection as open drainage as though the mere removal of a segment of rib made an opening into the pleural cavity. For various reasons the mortality gradually declined so that now we see reports averaging under 15 per cent by a number of surgeons. Two series stand out prominently, one by Whittemore (1919) of 100 cases with 6 deaths, and the other by Wynn (1921) of 56 cases with 4 deaths (7.1 per cent).

The influence of age has been appreciated for a long time. The statistics of Holt are quoted almost to the present. The mortality prior to 1913 in babies under two years was 67 per cent. Alexander and Sheik (1926) reporting a ten-year series, found the mortality to

be 30.5 per cent in the same age period and Douglas (1930) in a recent small series had a mortality of 15 per cent, exactly the same as my own experience.

TABLE I

SURGICAL DIVISION, HOSPITAL OF THE UNIVERSITY OF PENNSYLVANIA
EMPYEMA STATISTICS, SERVICE B, JULY, 1918, TO JULY, 1931

	MORTALITY BY AGES		
AGE	CASES	DEATHS	MORTALITY
0-10	49	2	4.1 per cent
11-20	14	0	0
21-30	14	0	0
31-40	12	2	16.7 per cent
41-50	5	0	0
51-60	2	1	50.0 per cent
61-70	3	1	33.1 per cent
Under 2 years	13	2	15.4 per cent
Under 14 years	52	2	3.9 per cent
Over 50 years	5	2	40.0 per cent
2-50 years	80	2	2.5 per cent

The high mortality of empyema in babies must be due to the fact that the effusion is large, resistance to infection not yet developed, intolerance to manipulation and sepsis from inadequate drainage by reason of timidity. The report of 33 cases with only two deaths by McEnery and Brennemann in babies treated by aspiration deserves serious consideration. Our own low mortality in children is due to the fact that most of them have come over from the pediatric service where for some years they have done one or more aspirations preliminary to drainage. Furthermore, in children, an opening in the chest during the pneumonia seems especially harmful and all recent writers urge that the drainage should be instituted during the metapneumonic stage. This statement holds true for all ages, and surgeons should be in no hurry to drain an empyema in the usual case until the pus thickens, indicating support and localization. However, this advice should be interpreted as delay in days and not weeks because undue delay produces fixed compression of the lung and the chronic cavity. A recovery from drainage of an empyema followed by death from thoracoplasty months later is a death for the patient from empyema.

The infecting organism is an important factor in mortality. The experience of the war time is known to all, but in civilian practice also the mortality of the streptococcic empyema is much greater than infection from the pneumococcus. Perhaps this statement bears some qualification because in the excellent study made by Locke (1930) while the mortality from the pneumococcic infection was 12 per cent as compared to the 20 per cent of the streptococcic, yet the mortality of the pneumococcus, Type 1, was 9 per cent and that of the other types aggregated 20 per cent. In our own series the organism was not typed. It is evident therefore that an important factor in the mortality is the organism causing the infection, a high percentage of streptococcic

cases will raise the mortality, and conversely a predominance of Type 1 pneumococcus will keep it low. In our series there were none of the diffuse streptococcic infections as seen in 1918-1919. In one of the fatal cases the pleural, peritoneal and pericardial cavities were involved but the pneumococcus was the infecting organism.

TABLE II

MORTALITY AND ORGANISM—99 CASES, 6 DEATHS, 6.1 PER CENT			
CULTURE	CASES	DEATHS	MORTALITY
Pneumococcus (45 per cent)	34	1	2.9 per cent
Streptococcus (24 per cent)	18	3	16.7 per cent
Staphylococcus (8 per cent)	6	1	16.7 per cent
Mixed	8	0	0
No Growth	10	0	0
Not Made	23	1	4.3 per cent

Another factor influencing mortality is that of complication. Middle ear disease, meningitis, bilateral empyema, pericarditis, etc., may kill the patient even though the pleura is perfectly drained. We had ten cases of middle ear infection but they were not a factor in the deaths.

Up to this point we have considered those factors influencing mortality which are based on what may be called the pathology of the disease; the influence of age, the state of the lung, the type of the infecting organism and the existence of complications. When the operation itself is considered one may be led into devious paths by the controversy between the advocates of the closed and so-called open drainage. I shall state my own opinion and present practice in the ordinary case.

Always in babies, usually in children, frequently in adults, one or more aspirations are done, especially if there is cyanosis, displacement of the heart or dyspnea. During the metapneumonic stage aspiration is followed by tube drainage introduced between the ribs, through a cannula, with local anesthesia, if the pus is thin and especially if the pleura is filled with fluid up to the apex. The tube is put into a bottle partly filled with water to get siphon drainage. The opening is kept air-tight with rubber dam. In a few days irrigations are begun with salt solution and sometimes Dakin's solution. The temperature subsides and progress is checked by x-ray. If drainage is not deemed satisfactory, a rib resection is done in two weeks and a larger tube is introduced. In 60 per cent this was necessary. When the patient has no respiratory embarrassment and but little heart displacement, the pus thick, and evidence of loculation apparent by x-ray, rib resection is done as a primary procedure. Aspiration may have preceded this. Forty-two per cent of the cases had primary rib resection. At this time I wish to make an important point. Resection of rib is performed simply to get more room for a larger tube. When the section of rib has been removed the chest is not open, a pair of curved hemostats is pushed through the intercostal muscles and pleura and followed immediately by the tube grasped in another pair of forceps. There is not sufficient

opening for a pneumothorax and the cavity is still full of pus. The muscles are grouped together about the tube with a stitch and the skin closed up. A suction apparatus is attached to the tube and some of the pus is removed, otherwise the patient is treated as described. I do not approve of an open drainage but closed drainage does not preclude rib resection. In the last seven cases we have used a Deryl Hart apparatus for tidal irrigation and suction and we are well pleased with the result especially as Overholt has devised a simpler modification.

TABLE III

TECHNIC MORTALITIES

	CASES	DEATHS	MORTALITY
Interrib puncture	23	2	8.7%
Interrib puncture (subsequent resection)	34 (60%)	1	2.9%
Total	57	3	5.3%
Primary rib resection	42	3	7.1%
	99	6	6.1%
	UNDER 10		OVER 10
Interrib puncture	19		4
Interrib puncture (plus resection)	13 (41%)		21 (84%)
	32		25
Primary resection	17 (35%)		25 (50%)
Total operations	49		50

Sick patients need not be moved from the bed but usually this is not necessary. I prefer local anesthesia but often we add nitrous oxide during the rib cutting. Dakin's solution is not used if there is evidence of a bronchial communication. This description represents our average procedure but of course interlobar collections, secondary pockets, etc., require methods adapted to the case. In the early cases in this series we did practice open drainage at times, and especially were apt to use two tubes, but for many years the method described has been used successfully as shown by the fact that since July, 1924, there has been a consecutive series of sixty cases without a death.

Drainage cannot be kept air-tight for more than a few days, but if a suction machine is used later this is immaterial and siphon drainage is practically as good as a suction. The drainage is continued until the cavity will hold less than two ounces when the tube is cut short, covered with an apron of rubber dam and gauze pad. The usual postoperative care is given every patient, breathing exercises, light treatments, high caloric diet, etc. As this paper is offered only for a discussion of mortality, the duration of drainage, late results, recurrence, etc., will not be discussed. I might add that two patients required minor thoracoplasty, several of the early cases were treated with bismuth paste, one case was

operated upon for bronchial fistula, one came back with the tube slipped back into the chest, and one case had osteitis of the rib ends requiring resection. This series represents the cases primarily operated upon by us. We had one patient operated upon by another surgeon ten years previous to a recurrent empyema as showing the necessity for end results to cover a long period of time. Brief details of the six fatal cases are as follows:

CASE I.—No. 60590. Female, aged sixty-nine years. No satisfactory history could be obtained. Was seen by a physician who immediately sent her to the University Hospital (Jan. 11, 1920). The patient was unkempt, dirty, and had bedsores from incontinence. She was quite toxic. The leucocytosis was 12,200. The left chest appeared solid with fluid, and aspiration withdrew 600 c.c. of turbid fluid containing *Staphylococcus aureus*. On the following day under local anesthesia a tube was pushed into the thorax between the sixth and seventh ribs and thereafter good drainage by the Kenyon method was effected. The urine output was low and the patient slowly declined in stupor until death occurred on the fourth day after admission.

Comment.—We do not see how we would alter the procedure in this case.

CASE II.—No. 60840. Male, aged thirty-five years, had empyema on admission to the University Hospital (Feb. 15, 1920) from a pneumonia which began eleven days previously. Aspiration (250 c.c.) revealed pus containing pneumococci. The leucocytes numbered 36,500. On the following day under local anesthesia, two rubber tubes were introduced through an interrib puncture. Drainage seemed satisfactory and irrigations with carbolic acid (1-60) were given. His general condition remained poor and signs of peritonitis and pericarditis appeared. He became more toxic and died ten days after operation. At autopsy the pleural cavity was found perfectly drained, the pericardium and peritoneum showing evidence of infection.

CASE III.—No. 63924. Male, aged thirty-six years. Following a streptococcic pneumonia, empyema developed, and patient was transferred from medical service, February 24, 1921. Under local anesthesia two rubber tubes were introduced through an interrib puncture. Much thick yellow pus was present. Drainage seemed satisfactory, but the patient remained ill. On March 3, blood culture heavily positive for hemolytic streptococci. On March 5, 30 c.c. of 1 per cent mercurochrome given intravenously followed by a chill. On the following day the fever was less, but again blood culture revealed heavy load of hemolytic streptococci. On March 8, mercurochrome repeated without reaction. Two days later patient died.

CASE IV.—No. 1812 N.S. Male, aged twenty-three months. Five weeks previously child developed pneumonia. Later had fever, chills, and sweats. X-ray showed fluid in left chest. Admitted Aug. 9, 1923. Aspiration below sixth rib posteriorly yielded pus at a depth of 4 cm., containing hemolytic streptococci. Rib resection done under nitrous oxide anesthesia and two rubber tubes inserted, open method. On August 11, antistreptococci serum given and improvement noted. Blood transfusion done on August 11 and again on August 14. Some improvement but on August 23 was worse again. X-ray showed lesion in upper lobe probably pneumonic. Died fourteen days after operation.

CASE V.—No. 2745 N.S. Female, aged thirteen months. Ten days previous to admission (Jan. 13, 1924) patient developed pneumonia. Crisis on ninth day followed by rise in temperature and prostration. On admission had dyspnea, cyanosis and a completely flat right chest. Abdomen distended. Temperature, 103.4° F., pulse 160. Under local anesthesia a small rubber tube was introduced into the

pleura by an interrib puncture. About 500 c.c. of pus were evacuated and the tube clamped. Following operation the temperature rose to 108, the pulse became rapid and feeble and death occurred six hours after operation.

CASE VI.—No. 3883 N.S. Male, aged fifty-five years. On June 25, 1924, had chill, pain in chest and "frictions." Admitted to medical service as a case of pneumonia. Fluid diagnosed on July 3. Aspiration revealed thin fluid smelling like b. coli infection but smear revealed streptococci. On this day under gas anesthesia an interrib puncture was done and drainage established. The tube was opened for ten minutes in every hour during the first six hours. Respiratory rate very high. On July 8 he was still toxic with fever, sweats and cyanosis and under gas anesthesia a rib resection was done and considerable pus evacuated and the cavity drained. But little improvement was manifest and as the x-ray showed incomplete drainage, the cavity was explored on July 15 under local anesthesia. A pus pocket containing 100 c.c. of pus was found and drained. He died the following day (July 17, 1924).

SUMMARY

1. A series of 99 cases of acute empyema following pneumonia with six deaths (6.1 per cent) is reported.

2. The death rate in children under two years was 15.4 per cent; in adults over 50 it was 40 per cent. In children under 14 years it was 3.9 per cent.

3. Mortality is largely affected by the age, state of the lung, infecting organism, and infective complications.

4. Except for aspiration, thoracotomy should be delayed until the metapneumonic stage.

5. Drainage in many cases can be effected by interrib tube drainage. In others rib resection may be necessary.

6. Rib resection is not open drainage if properly managed.

7. Closed drainage is the proper method in nearly all cases and will effect a cure in nearly every case.

DISCUSSION

DR. E. F. BUTLER.—I have recently reviewed the statistics of 323 cases of empyema in my own experience. The mortality figures were quite similar to those reported by Dr. Muller—10.7 per cent for acute pneumococcus empyema—21.6 per cent for acute streptococcus cases—a much higher percentage for mixed infections following abdominal conditions, lung abscess, etc.

Age was a definite factor in the mortality figures. In acute pneumococcus empyema during the first five years of life the rate was 10 per cent. During the second five years of life it dropped to 5.5 per cent. From age ten to age forty the mortality was 7.8 per cent. After age forty it rose to 36.5 per cent.

I have relied upon aspiration during the formative or synpneumonic phases of empyema and postponed operation until the metapneumonic phase had been reached. In approximately one-half of the cases closed drainage has been employed in the primary attack, but has always been followed by secondary open operation. Of three cases in which open operation was employed in the primary attack, temporary wound occlusion became necessary. There was no mortality in this group. On 13

occasions I have utilized the type of operation described by Dr. Pickhart in 1923. There was no mortality in this series and my experience with the operation has been quite satisfactory.

DR. D. S. ALLEN.—The treatment of empyema in children presents many problems. There are those who advocate prolonged aspiration, those who advocate drainage with the wound closed about a rubber tube and those who do not make an attempt at closure of the wound in the chest wall. In the latter instance the empyema cavity is opened widely and drainage carried out much as in abscesses elsewhere in the body.

In a consideration of the results of the various types of treatment for acute empyema in children we should be very careful in defining exactly which are really empyemas and which are not. An empyema is not correctly defined as such until there is actual pus in the chest. We all see fluid in the chest of babies which is contaminated by bacteria and leucocytes but which is not actually pus. If such cases are aspirated they often recover. Sometimes a single aspiration will suffice. Other cases go on to the formation of actual pus and in these cases continued aspiration alone may not be the best treatment.

There are two things we strive to do in the treatment of acute empyema in children. First of all, we try to save life. If external drainage is deferred until there is thick pus in the empyema, the mortality will be lower than if drainage is instituted while the pus is still thin.

The second feature to bear in mind in these children is to have them recover as quickly as possible. If aspiration alone is carried out after the pus becomes thick the disease is prolonged. At the St. Louis Children's Hospital drainage is instituted after the pus has become thick. I do not think it makes a great deal of difference whether the tube is kept closed or wide open, so long as adequate external drainage is maintained.

We have recently gone over our results at the St. Louis Children's Hospital tabulating a series of 112 cases operated upon between the years of 1925 and 1930. The plan of treatment in these cases was to carry out aspiration until the pus became thick and then open drainage was instituted. The empyema cavity was opened, usually with rib resection. There were 32 children under two years of age with four deaths. In all four of these deaths there were complications, such as meningitis and scarlet fever. One case developed scarlet fever after the empyema had healed. There were 80 cases over two years of age with five deaths, a mortality of 6.2 per cent in this group. The entire group showed a mortality of 7.1 per cent.

These results compare favorably with other types of drainage. Whether the tube is closed or open therefore does not seem to affect the mortality greatly. The important thing is to institute adequate external drainage at the proper time.

DR. F. BOLAND.—I was particularly interested in hearing Dr. Muller state that the mortality in acute empyema was not so dependent upon the method of operation but upon virulence of the infectious organism. I believe that is the thing which accounted for a great many of the deaths in 1918. It has been the custom to ascribe the high mortality of 40 or 50 per cent in the army camps to the method of operating. But I feel sure that in the cases I saw at Camp Gordon, probably one hundred and fifty, which showed a mortality of 40 per cent, a great many were doomed from the beginning, regardless of what operation was done, due to the virulence of the organism, the streptococcus hemolyticus. I don't believe any method of operation would have saved some of those soldiers. I think it is a fair point to bring out, not always to blame the method of operation for the high mortality in empyema in our army camps.

DR. L. ELOESSER.—Dr. Robinson has goaded me to activity.

There was a Scotch doctor in this town, Dr. Shields, who used to say there were two kinds of pus in empyemas. The one was the "My God" kind and other the "Thank God" kind of pus. By that he meant the thin, foul pus was the "My God" kind, and the other was the "Thank God" kind.

As for the operation: The position of the patient on the operating table is of much importance. If he is placed on his good side there is a sudden inrush of air after the rib is resected; there is a sudden collapse of the lung; and, when the patient coughs, a flapping back and forth of the pneumonic lung which must be and is provocative of harm.

On the other hand, if you will sit the patient up, or if he is not able to sit, leave him on his bad side and pull him to the edge of the table, and working from below resect a rib and introduce a valved drainage tube, the pus will flow out by gravity, no air will enter, and the distressing violent distention and collapse of the lung will be avoided. We often operate upon patients in bed, either in sitting them up, or pulling them over to the edge of the bed.

I should like to ask Dr. Muller what he means by closed drainage, for, in the end, almost all of our drainages are closed drainages. When you get a dressing soaked with pus and moisture over the drain, then the open drainage becomes a closed one.

As far as the apparatus goes, I think that the more complicated it is the more liable it is to get plugged with pus and fibrin, and to act as a plug rather than a drain.

DR. W. E. LEE.—There is just one phase of Dr. Muller's paper that I would like to emphasize, and that is the necessity for differentiating the surgical treatment of empyema in children from that in adults. I recall very vividly my early experience as one of the younger surgeons in the Children's Hospital, and my first emergency operation, which was for massive empyema in an infant. Looking over the instruments, I failed to find a rib resector, and the nurse's reply was very curt and pointed—"You certainly are not going to resect the rib in a two-year-old infant." When I insisted, she replied, "The child will die." This did not convince me, I went ahead and resected the rib and the child did die. In a short time there was another case about the same age and this time the nurse asked me if I wanted a rib resector and I again insisted. I resected the rib and this child died. This has been a great many years ago, but the tradition at the Children's Hospital began long before my time, and this experience of many years has certainly demonstrated the unusual danger in performing immediate and open thoracotomy for empyema in infants. We did not know the explanation at that time, but the clear-cut, convincing research of Evarts Graham has made it all clear, and for me removes from the field of discussion the question of immediate and open thoracotomy in the treatment of empyema in infants.

DR. G. P. MULLER.—In this brief paper I only intended to bring out some important points. The first is that mortality is dependent upon the pathology of the lung and certain complicating influences, provided that drainage has been satisfactorily done.

The other point is that the mere fact that a piece of rib is resected should not be used as an indication that the operation consisted of open drainage.

THE DIAGNOSIS AND TREATMENT OF DIAPHRAGMATIC HERNIA*

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THE diagnosis and treatment of diaphragmatic hernia is of equal interest to the clinician and the surgeon. The diagnosis is of importance to the clinician because the symptoms are often obscure, and because of the frequency with which the condition must be considered in the differential diagnosis of complicated abdominal and thoracic conditions. The treatment is of primary concern to the surgeon for operative replacement of the herniated viscera, and repair of the abnormal opening in the diaphragm is the only treatment that promises complete relief of symptoms.

The incidence of diaphragmatic hernia is probably no greater now than it was twenty years ago; at that time it was thought to be uncommon, and its recognition in life was extremely rare. More frequent recognition of the condition in the last two decades has been due primarily to development of roentgenologic methods of diagnosis. The clinical study of proved cases has established a fairly definite symptomatology which has enabled the clinician to diagnose or suspect the presence of a hernia and to have a special roentgenologic examination made. I do not believe that the entire credit for the present more frequent recognition of this condition is due to the roentgenologist, but that he must share this credit with the clinician. A greater proportion of the responsibility for the establishment of the correct diagnosis must rest on the clinician, for he is first to come in contact with the patient, and the subsequent course of the examination and treatment depend on his interpretation of symptoms.

The recognition of diaphragmatic hernia from the clinical symptoms only is often difficult, because the complex symptoms often simulate those of other diseases of the abdomen and thorax of which the most common are cholecystitis, gastric ulcer, cardiospasm, intestinal obstruction, and angina pectoris. I shall not go into details of differential diagnosis, but shall give an outline of the usual symptoms associated with diaphragmatic hernia. The symptoms are often complex because of the various structures involved in the hernia. They depend on the amount of mechanical interference with the function of the herniated abdominal viscera, on the degree of interference with the normal function of the diaphragm, and on the amount of increased pressure within

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the thorax, which causes impairment of respiration and circulation. These conditions, which influence the syndrome of hernia, depend, to some extent, on the type of hernia present, whether congenital or acquired, and whether or not trauma was an etiologic factor.

Inasmuch as the embryologic development of the diaphragm plays such an important part in the susceptibility, occurrence, and classification of diaphragmatic hernia, I shall give a brief summary of its formation. This is a greatly involved process, for the muscular elements of the diaphragm are derived from several sources.

The anterior, lateral and central parts, which comprise the greater portion of the diaphragm in the adult, are formed from the transverse septum and the fused ventral mesentery. The remaining, posterolateral portion is formed by fusion of the dorsal mesentery, the mesoderm that is derived from the receding wolffian body, and the pleuroperitoneal membrane that is derived from the pulmonary ridge. It is difficult to determine the exact amount of muscle that is derived from each of these structures, for there is probably considerable variation. However, it is probable that the dorsal mesentery forms the posterior and central portions, containing the esophageal opening. The mesodermal cells, from the receding wolffian body, form the right and left crura. The pleuroperitoneal membrane grows ventrally, closes the remaining opening (hiatus pleuroperitonalis) between the peritoneal celom and pleural celom by fusing with the transverse septum, and forms the lateral portion of the diaphragm. Failure of fusion, or of proper deposition of the mesoderm at any one of these adjacent points of union may result in congenital continuity of the pleural and peritoneal cavities or in a congenitally weak portion of the diaphragm. Consequently, from an embryologic standpoint, weak portions might be expected to appear at the point of fusion of these different structures. These portions are situated dorsolaterally, at the fissure pleuroperitonalis (foramen of Bochdalek); also at the outer crus and at the esophageal opening. Hernia through the dome is common, but it cannot be explained on the basis just given, for it is not an area where fusion takes place. It may be the result of excessive degeneration of muscle in the formation of the central tendon, or of some pathologic condition. Unilateral absence of the diaphragm is probably the result of failure of development of the pleuroperitoneal membrane which usually is found as a narrow ridge of tissue along the posterior wall of the thorax.

CLASSIFICATION

There are numerous classifications of diaphragmatic hernia based on embryology, etiology, pathologic anatomy, the site of the opening in the diaphragm, the presence or absence of a sac, the content of the hernia, and other conditions. It is difficult or impossible to make most of these

classifications clinically, and many of them are of little practical value. They are usually classified in three main groups: (1) congenital, (2) acquired and (3) traumatic.

The group of congenital hernia, as usually thought of, includes only those cases in which the hernia is present at birth and results from a developmental defect in the diaphragm. In these cases, there are often developmental defects in other parts of the body. The majority of the infants who have these defects die soon after birth. It is possible that the diaphragm may be normal at birth, and that a hernia may be produced immediately after birth by the institution of forceful methods to establish respiration. These hernias may be considered congenital, or if the injury does not cause a complete opening through the diaphragm, a potential hernia remains, and may cause symptoms later in life.

In the group of acquired hernia, most writers include all cases that occur after birth, and may be due to any condition which increases intraabdominal pressure. In these cases, the hernia usually occurs at a point of embryologic fusion of the diaphragm. The most common hernia of this type is that which occurs through an enlarged esophageal hiatus commonly called esophageal hernia and this is the most common hernia seen in adult life. In these cases there may or may not be a history of injury, and often it is difficult to associate a given injury with the onset of symptoms.

The group of traumatic hernia is limited by certain writers to those cases in which there has been direct laceration of the muscle of the diaphragm, as by gunshot or stab wounds, or by rupture of subdiaphragmatic abscess. However, from a practical standpoint, I believe that all cases in which the onset of symptoms follows injury, should be placed in this group, regardless of whether the injury to the diaphragm was due to direct or to indirect force. In the cases of direct injury, the opening in the diaphragm may be situated at any point. In the cases of indirect injury, such as crushing injuries from falls or automobile accidents, the opening may be at a point of fusion of the diaphragm, as in the acquired type, or at any point. The most common situations in such cases are the dome and posterior half of the diaphragm; in some instances the muscle is torn from its attachment to the thoracic wall.

From a clinical and surgical standpoint the history of a preceding injury is helpful in establishing the diagnosis and in determining the type, urgency, and prognosis of the operative treatment. Because of this practical clinical and surgical significance of trauma as an etiologic factor, I have suggested the following classification of diaphragmatic hernia into two main groups, nontraumatic and traumatic, and have subdivided these according to the various types.

Nontraumatic diaphragmatic hernia.

1. Congenital, due to embryologic deficiency, usually without an enclosing sac. The most common sites in probable order of frequency of occurrence are: (1) through the hiatus pleuroperitonalis (foramen of Bochdalek), (2) through the dome of the diaphragm, (3) through the esophageal opening, (4) through the foramen Morgagni and (5) through the gap left by absence of the left half of the diaphragm.

2. Acquired after birth. The sites of occurrence are: (1) through a point of embryologic fusion of the diaphragm; (2) at sites named under congenital types; (3) through the esophageal hiatus. The type last named has an enclosing sac.

Traumatic diaphragmatic hernia.

1. Indirect injury to diaphragm. It may occur at any point, including points of embryologic fusion, but the most common region is the dome and posterior half of the left side of the diaphragm. It usually is the result of a severe, crushing injury and may or may not have a sac. When the hernia is through the esophageal opening there is a sac, and when through the leaf of the diaphragm, there usually is no sac.

2. Direct injury to the diaphragm. The hernia may occur at any point, and is usually the result of penetrating wounds, such as those from a gunshot or a knife. Rupture of subdiaphragmatic abscess or empyema may be the cause. Usually there is no sac.

The presence or absence of an enclosing hernial sac is of some practical clinical and surgical importance. A sac is usually not present in congenital hernias where there has been loss of structure of the diaphragm, or in traumatic hernias due to direct or indirect injury. These may be said to be not true hernias, but instances of evisceration of the abdominal organs. Usually several abdominal viscera, and a large portion of each involved organ are concerned, and symptoms are very severe and urgent. In surgical repair of these hernias, preliminary phrenicotomy is often advisable and intratracheal anesthesia should be used during the repair. Hernias with a limiting sac are considered true hernias, and rarely involve more than one organ. The most common hernia of this type is the esophageal hernia, in which the stomach is usually the only abdominal organ involved. The symptoms in these cases are more uniform than in others, and in the operative repair of the hernia, the sac must be either entirely removed or its continuity with the abdominal cavity must be obliterated.

In the tabulation the clinical and operative observations and results are based on forty-five cases of diaphragmatic hernia in which I have operated in the last five years.

The syndrome in the sorts of cases included in the tabulation can be divided into two groups, the esophageal type in which the stomach only is incorporated in the hernia and the traumatic type, in which the stomach, large and small bowel are involved in the hernia.

Esophageal diaphragmatic hernia may be considered to be congenital, acquired or traumatic, but it is essentially congenital. The symptoms may begin at birth or at any time during life. There may be a congenital weakness in the esophageal ring, and a definite hernia may later be produced by some type of injury or by increased intraabdominal pressure. These cases may be considered of traumatic origin but are essentially congenital, in the same manner as hernias in the inguinal region are congenital. The esophageal type of hernia produces more uniform symptoms than hernias elsewhere in the diaphragm, because the

Fig. 1.—Herniation of about half of the stomach into the left side of the thorax through the esophageal hiatus.

stomach is usually the only abdominal organ involved. The symptoms are those of intermittent or progressive incarceration and obstruction of the stomach. At the onset, the attacks are usually mild; they consist of epigastric distress that is projected through to the back, come on during or shortly after a heavy meal, and are relieved by belching of gas or by vomiting. These attacks are commonly considered to be due to, and the patient is treated for, cholecystitis. As more of the stomach becomes incorporated in the hernia, the attacks become more severe; the pain is projected straight through to the back and to the lower left side of the thorax, is more marked to the left of the spinal

column, and often appears between the shoulder blades. This pain is agonizing, and there is difficulty in belching of gas and vomiting because of spasm of the diaphragm and reflex cardiospasm. The spasm of the diaphragm produces an hour-glass deformity of the stomach, which interferes with the emptying of the upper loculus, and causes increased intragastric pressure. The pressure of the herniated portion of the stomach on the lower part of the esophagus interferes with belching of gas or vomiting. Spasm of the diaphragm is commonly associated with phrenic pain that is referred to the left shoulder and which at times may radiate down the arm. The increased pressure within the thorax causes cardiac embarrassment, with palpitation and tachycardia. Pressure on the lung and interference with the motion of the diaphragm cause dyspnea. These symptoms are augmented when the patient lies down, and in the more severe cases it is necessary for patients to sit up to breathe. The attacks may last for from a few minutes to several hours, and occasionally are considered to be caused by myocardial disease. The attacks usually are completely relieved by vomiting and often recur immediately after food is taken. There is often an interval of weeks or months between attacks. It is probable that during the interval between attacks the stomach is not incorporated in the hernial ring, and is in its normal position below the diaphragm. When the attacks become more or less constant, the stomach usually has become fixed in the thorax by adhesions. All of the early symptoms of pressure are augmented during the attacks. There is loss of weight from inability to retain food and from marked restriction in diet due to fear of bringing on an acute attack. This may be termed "food-fear." The vomiting is more severe during attacks and often is of the retention type. During the severe vomiting, the vomitus may contain blood. If the attacks are of long standing, the patient not uncommonly has a burning sensation in the epigastrium after meals, which is relieved by taking small quantities of food. If large amounts of food are taken, it may bring on one of the attacks that is associated with incarceration of the stomach. Several of these patients have been given a diet for ulcer, and have obtained partial relief because they have taken a restricted amount of food at frequent intervals. Hemorrhage is not a common symptom; it usually is indicative of severe incarceration, with fixation of the stomach in the thorax. The bleeding is caused by erosion of the mucous membrane, due to the forceful pressure exerted during the attacks of vomiting on the large, distorted, congested, and fixed stomach. This erosion may be superficial, or, in cases of long standing, may form definite ulceration from repeated trauma. This is usually the final stage of incarceration. I have never seen a stomach which was strangulated as a result of hernia nor do I believe that it is possible because of the powerful musculature and rich blood supply in the gastric wall. Due to the fixation of the stomach in the thorax the ob-

struction may be complete, and the vomiting may be so severe as to produce gastric tetany (Figs. 1, 2, 3 and 4).

The foregoing is the clinical picture of a typical case of slowly progressive herniation of the stomach through the esophageal hiatus, but, of course, it cannot be expected that every case will present all the symptoms, any more than it can be expected that a typical history will be elicited in every case of any other organic condition. The chief symptoms depend, to some extent, on the time in the course of the disease at which the patient is examined, on the rapidity with which

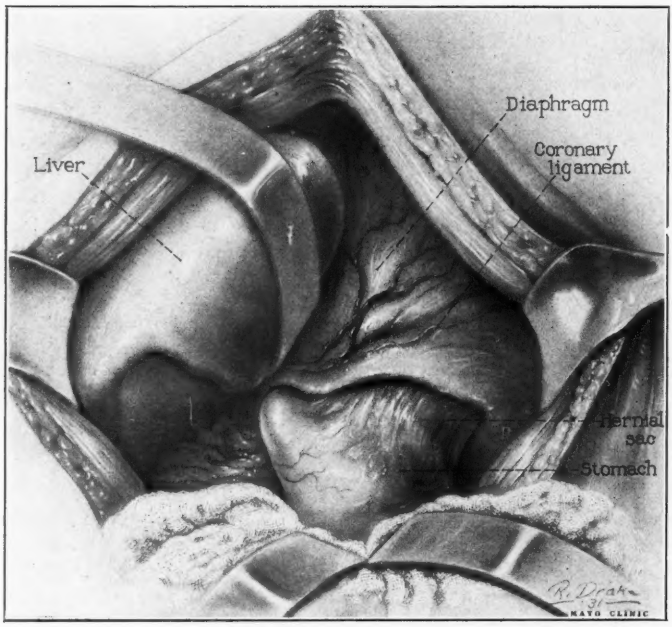

Fig. 2.—Exposure of the upper margin of the esophageal opening in the diaphragm by cutting the coronary ligament of the left lobe of the liver.

the hernia has been produced, on the amount of fixation of stomach in the thorax, and on the amount of disturbed function of thoracic organs.

Among the forty-five patients previously mentioned, the age incidence was as follows: among the young patients, one each was aged seven months, three years and twelve years; four patients were in the decade of life between twenty and thirty years; six, between thirty and forty years; eleven, between forty and fifty years; twelve, between fifty and sixty years; eight between sixty and seventy years, and one patient was seventy years of age. Twenty-three patients were females; twenty-two, males. The duration of symptoms was from five weeks to twenty-four years; the average duration for the group of forty-five was seven years.

The chief complaints and symptoms occurred in the following frequency: epigastric pain in thirty-six cases; vomiting in nineteen; dyspepsia in seventeen; hemoptysis in eleven; difficulty in swallowing in seven; weakness in seven; palpitation of the heart in three; and paralysis of the legs and gastric tetany in one case each.

The situation of pain was as follows: in the epigastrium in thirty-six cases; in the precordium in three; behind the sternum in twelve; referred directly to the back, from the primary site of pain, in nine; projected to the left side of the thorax, from the primary site of pain, in thirteen; and to the left shoulder in sixteen.

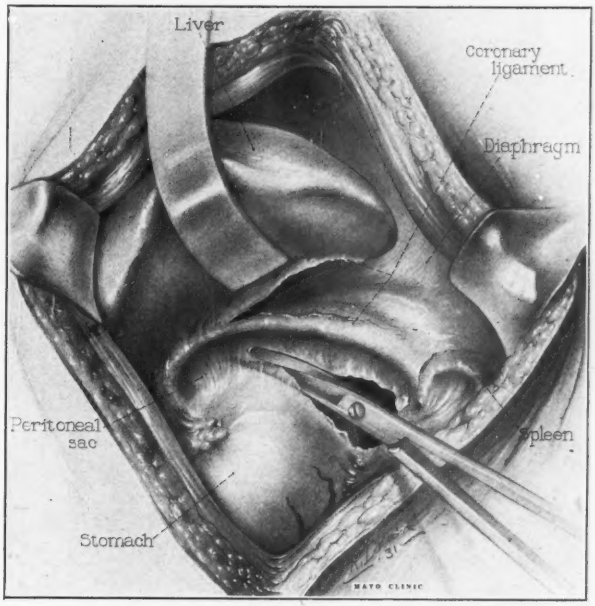

Fig. 3.—Removal of the sac from its attachment to the stomach and from the enlarged esophageal hernial opening.

Measures which brought relief included the following: vomiting in twenty-three cases; taking of soda in eight; belching of gas in six; taking of food in three; administration of morphine in three; administration of other sedatives in three; lying down in one case, and administration of an enema in one.

The symptoms in traumatic cases in which the stomach only is involved are practically the same as those in which the hernia is through the esophageal opening, but in most of the traumatic cases the colon and small bowel are involved, and there are the added symptoms caused by interference with the function of these structures. This may be indicated by obstinate constipation, with accumulation of large quanti-

ties of gas in the colon, or by periodic attacks of partial or complete obstruction. The sudden onset of symptoms in traumatic cases is usually directly related to the injury, the progress of symptoms is much more rapid than in esophageal cases, and the first symptoms may be those of severe gastric hemorrhage or of intestinal obstruction. The respiratory symptoms are usually more marked in traumatic than in esophageal cases, because of the greater amount of viscera contained in the thorax. These viscera are in direct contact with the pleura and pericardium, for there is no hernial sac (Figs. 5, 6, 7, 8 and 9).

Fig. 4.—The stomach two months after abdominal repair of diaphragmatic hernia. The entire stomach is below the diaphragm.

Because of the frequency with which the symptoms of diaphragmatic hernia may simulate other organic disease, I have reviewed the entire group of forty-five cases to determine what the patients had been treated for before the correct diagnosis had been established. In fourteen cases, diagnosis and treatment had been that of cholecystitis, and seven of the patients had been operated on. In thirteen cases, diagnosis and treatment had been that of gastric or duodenal ulcer, and one of the patients had been operated on. Three patients had been treated for intestinal obstruction and one patient had been operated on. In four cases the diagnosis had been heart disease, and treatment had been given accordingly.

TREATMENT

In cases of esophageal diaphragmatic hernia, in which the stomach only is involved, and in which the symptoms are mild, the treatment may be conservative. If symptoms become progressively more severe, the possibility of serious complications is great, and I believe that treatment in all such cases should be surgical unless radical operation is contraindicated because of the patient's general condition. Operation should be performed before severe incarceration, with obstruction and traumatic lesions of the stomach, have occurred. The operative risk is

Fig. 5.—The entire stomach is in the left thoracic cavity. Partial collapse of the left lung.

increased by gastric retention and the technical difficulties are enhanced by fixation of the stomach to the diaphragm and to structures within the thorax.

All cases in which the colon or small bowel are involved in the hernia demand early operation because of the danger of intestinal obstruction. However, these cases are usually traumatic in origin and it is best not to operate until the acute symptoms due to the injury have subsided, if the patient's condition will permit.

Preliminary paralysis of the diaphragm, produced by phrenicotomy, is often of value in incarcerated and strangulated hernias, because it pre-

vents spasm of the muscle and relaxes the hernial ring. It is of great advantage in closing large hernial openings, when there is considerable loss of structure of the diaphragm, for relaxation of the muscle permits the defect to be closed without tension, and if the diaphragm has been torn from the wall of the thorax it can be sutured to the intercostal muscles. Phrenicotomy is also of value in cases in which there is moderate congenital shortening of the esophagus; the relaxation and elevation of the diaphragm following this procedure permits the hernial opening to be sutured around the lower part of the esophagus, entirely

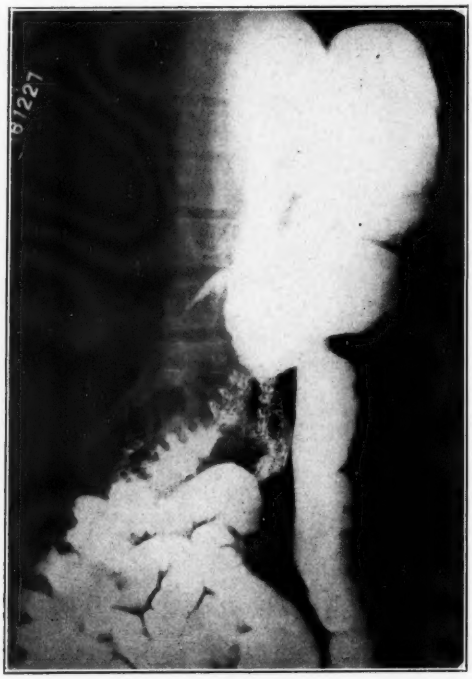

Fig. 6.—Herniation of entire stomach, greater portion of transverse colon and splenic flexure of colon into the left thoracic cavity.

above the herniated portion of the stomach. Approximation of small hernial openings, usually the esophageal type, can be accomplished without undue tension, and phrenicotomy is not necessary. In some instances in which it seems advisable to reestablish the function of the diaphragm, the nerve may be crushed or cut, and then sutured, and function will be reestablished within three to six months thereafter.

Phrenicotomy may be used as a palliative measure when the radical operative procedure of closure of the defect in the diaphragm is contraindicated because of the patient's condition. The purpose of this procedure is to prevent spasm of the diaphragm, which is the cause of

the severe attacks of incarceration of the stomach. I have performed this procedure in four cases in which radical operation was contraindicated because of the general condition of the patient. Two patients were more than seventy years of age with myocarditis and poor cardiac compensation. One patient came to the clinic primarily for renal colic. It was necessary to remove one kidney, and the function of the remaining kidney was not good. The symptoms of one patient, an infant, were markedly relieved following phrenicotomy and the parents did not wish the child to be submitted to radical surgery. This procedure does not completely relieve the symptoms. There is always a moderate amount of gastric distress immediately after, or shortly after, heavy meals, but the patients get along fairly well if they are careful with their diet.

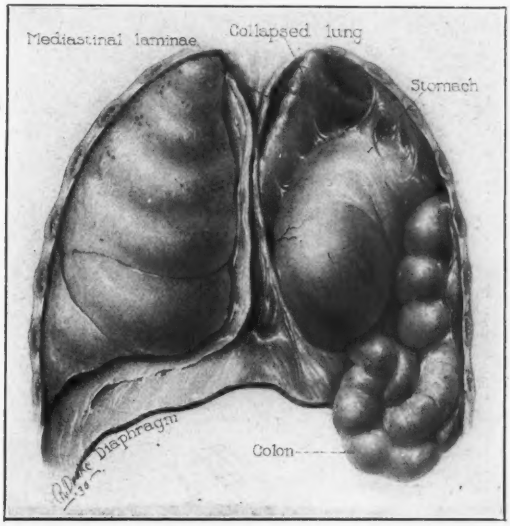

Fig. 7.—Adhesions of the herniated stomach and colon to the lung and thoracic wall as high as the second rib. Drawing made at the time of operation.

Phrenicotomy relieves the acute attacks of incarceration if the stomach is not fixed in the thorax. It is not applicable to hernias in which a large portion of the stomach is in the thorax, causing marked pressure on the heart and lungs, nor is it applicable in any case in which the intestines are involved in the hernia. This procedure should not be employed when radical operative repair can be effected.

The only operative procedure which will assure complete relief of symptoms is replacement of the herniated abdominal viscera and repair of the abnormal opening in the diaphragm.

I have used both the abdominal and the thoracic approach in the reduction and repair of these hernias. In two cases in which the thoracic approach was used it was necessary to resort to a combined thoracic

and abdominal procedure in order to free the adherent abdominal viscera from the abdominal side of the diaphragm, if repair was to be accomplished without injury to abdominal viscera. In one of these cases postoperative empyema developed, necessitating drainage and resection of a rib before ultimate recovery. I prefer the abdominal approach because I believe there is less risk of thoracic complications. This approach is of particular advantage in cases of esophageal hernia, for the herniated stomach is usually confined in a sac in the posterior mediastinum, and does not enter the true pleural cavity.

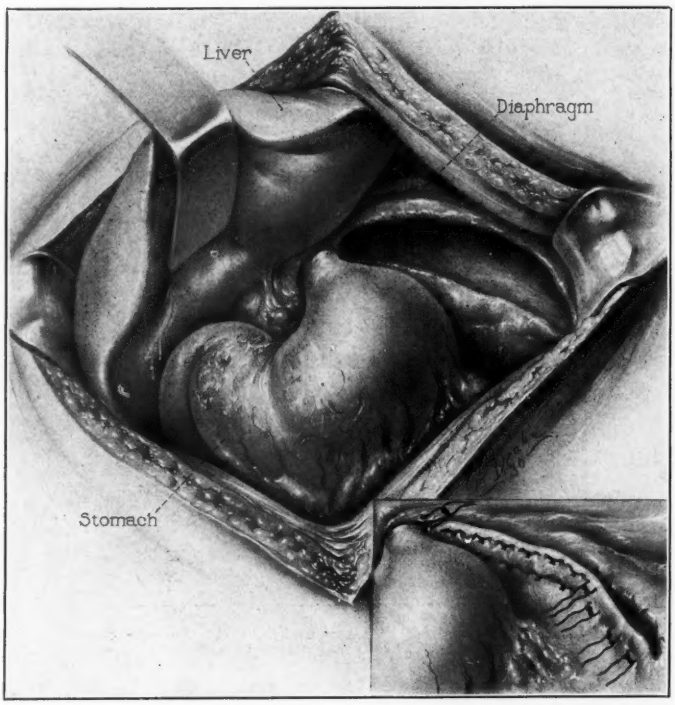

Fig. 8.—Drawing made after replacement of the herniated viscera into the abdomen. A large rent is shown extending entirely across the posterior two-thirds of the diaphragm. The insert shows the closure of the hernial opening by overlapping the muscle of the diaphragm; this is facilitated by a preliminary paralysis of the muscle by phrenicotomy.

The technical difficulties of adequate exposure in these cases are often difficult because fixation of the left lobe of the liver to the diaphragm by the coronary ligament makes the esophageal opening inaccessible. Exposure of the upper margin of the opening is greatly facilitated by cutting this coronary ligament and retracting the left lobe of the liver to the right. The spleen is often very adherent to the lower margin of the opening and posterior part of the diaphragm, and usually can

be separated by blunt dissection and retracted posteriorly by a properly constructed retractor. The sac of the hernia in these cases must be either completely removed or cut from its attachment to the stomach and hernial opening before the enlarged esophageal hiatus is repaired. It is important to place a stomach tube through the esophagus into the stomach before closing the opening around the lower part of the esophagus; this prevents constriction due to tight closure. In traumatic cases of long standing, the abdominal viscera are usually adherent to the structures in the thorax, often as high as the apex of the lung.

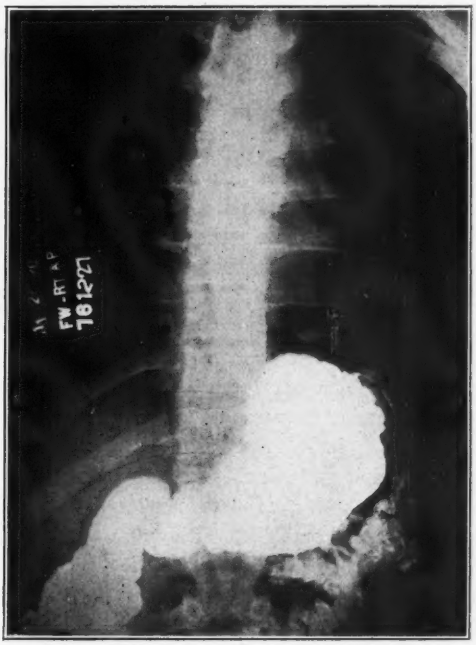

Fig. 9.—Stomach and colon is shown in normal position below the diaphragm one month after abdominal repair of diaphragmatic hernia.

These adhesions can be separated by approaching them through the hernial ring, with less danger of injury to the abdominal or thoracic viscera than if another approach is used, for the definite relationship of the herniated structures can be established. In many instances it is necessary to ligate, or otherwise to prevent bleeding from, the bands of adhesions high in the pleural cavity, either on the visceral or parietal surface. This is often satisfactorily accomplished with the high frequency coagulation cautery when it is difficult to tie a ligature.

The abdominal approach is of value in determining the presence of associated lesions in the herniated abdominal viscera as well as any

other pathologic lesion within the abdomen which may require treatment at the time of operation or later. The associated conditions which were present in the thirty-eight cases of this series, in which the abdominal approach was employed in repair of the hernia, were as follows: In eight cases there was traumatic gastric erosion; in two cases, definite gastric ulcer; in two cases, duodenal ulcer; in four cases, cholelithiasis, and in one case, carcinoma of the esophagus.

The operative procedures in the forty-five cases here reported are as follows: In four cases palliative left phrenicotomy was done, in three cases phrenicotomy was done preliminary to operative repair, but for some unavoidable reason, the main operation was delayed. In the remaining thirty-eight cases the herniated abdominal viscera were replaced in the abdomen, and the abnormal opening in the diaphragm was repaired. In two cases a combined thoracic and abdominal approach was made. In thirty-six cases, the abdominal approach was made. In one case, a Polya type of gastric resection was done for gastric ulcer high on the lesser curvature of the stomach at the time of operation. In one case, appendicostomy was done at the time of operation because of marked dilatation of the colon resulting from partial obstruction during its residence in the thoracic cavity. In three cases there was moderate congenital shortening of the esophagus, associated with the hernia. The diaphragm could be sutured entirely above the stomach after the diaphragmatic muscle had been paralyzed by phrenicotomy. In seventeen cases, preliminary phrenicotomy was done, and in one case, preliminary phrenicotomy and extrapleural thoracoplasty were performed. There were five deaths following operation. One patient, who had a congenital type of hernia through the hiatus pleuroperitonalis, died on the second day, from shock or cerebral embolism, although a definite cause of death was not found. One patient with a traumatic type of hernia died on the seventh day from pneumonia involving the right lung, with partial collapse of the left lung; he was *in extremis* at the time of operation and had been unable to retain food or lie down to sleep since the accident six weeks before. In three of the fatal cases the hernias were of the acquired esophageal type; one patient died on the twelfth day from pulmonary embolism; one on the second day, from respiratory and cardiac failure, and one, three hours after operation from respiratory and cardiac failure for which definite cause was not found. The latter two patients were very obese, and although operation was delayed several weeks to give time for the patients to reduce in weight, they could not reduce without sacrificing strength. I believe reduction in weight is very important in these cases, particularly if a large portion of the abdominal viscera has been in the thorax for a long period. In these cases, the herniated viscera have lost their right of residence in the abdomen, and reduction of weight is necessary before their replace-

DATA CONCERNING SITUATION, ETIOLOGY, AND CONTENT OF HERNIA IN FORTY-FIVE CASES*

SITE OF OPENING	CASES	ETIOLOGY	CASES	CONTENTS OF HERNIA	CASES
Esophageal hiatus	31	Congenital	5	Stomach only	28
		Acquired (history of trauma, 10)	23	Stomach only	2
		Congenital short esophagus	3	Stomach and colon	1
Hiatus pleuroperitonalis	1	Congenital	1	Colon and small bowel	1
Posterior half and dome of diaphragm	13	Trauma:		Stomach only	2
		Direct injury	3	Stomach and colon	4
		Indirect injury	10	Stomach, colon, and small bowel	1
				Stomach, colon and spleen	2
				Stomach, colon, spleen and small bowel	4
Total	45		45		45

*In all forty-five cases, the hernia was through the left side of the diaphragm; in six, there was encroachment in the right side of the thorax.

ment, because of the danger associated with the increased pressure when replacement has been effected.

I have recently reviewed replies to letters of inquiry that were sent to the thirty-nine patients who recovered. Of the three patients on whom palliative phrenicotomy was done, one has since died of angina pectoris and the two others have obtained partial relief of symptoms. Of the thirty-three patients for whom radical operative procedures were done all have received relief of symptoms, except for a few minor complaints, and there have been no recurrences. All patients have been reexamined six months to one year, or every year, after operation.

SUMMARY

Diaphragmatic hernia is relatively common. The symptoms of diaphragmatic hernia are usually definite, and depend on the amount of mechanical interference with function of the organs involved. Diagnosis from clinical symptoms only is often difficult, because the syndrome often simulates that of other organic diseases, the most common of which are cholecystitis, gastric ulcer and heart disease. Roentgenologic examination of the stomach is important in establishing a definite diagnosis. Operative replacement of the herniated viscera into the abdomen, and repair of the abnormal opening in the diaphragm, is the only treatment which insures complete relief of symptoms. Palliative phrenicotomy may be used in selected cases in which operation is contraindicated. Preliminary phrenicotomy is often of value in repair of enlarged hernial openings if there has been considerable loss of structure. Of the forty-five surgical cases here reported, radical operation was performed in thirty-eight. There were five operative deaths and no recurrences.

DISCUSSION

The discussion of this paper will be found with that of Dr. Eggers' at the end of Dr. Eggers' paper, p. 48.

HERNIA OR EVENTRATION OF THE DIAPHRAGM
REPORT OF A CASE*

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THE difficulties connected with the correct diagnosis in a recently observed patient is the excuse for the presentation of this subject. It is usually believed that the two conditions are so well known and the pathology of each so well understood that there should be no difficulty in making a differential diagnosis. In the patient whose history is presented herewith, however, that did not prove to be the case.

A female infant, N. H., four weeks old, was admitted to the Lenox Hill Hospital, March 6, 1931. She was referred by Dr. Percy C. Fisher of Ridgewood, N. J., on account of a diaphragmatic hernia. She was the first child of normal, healthy parents, and had been delivered with forceps at full term after a long labor. During the first twenty-four hours after birth the baby seemed all right, but then with each attempt at breast feeding it became alarmingly short of breath and cyanotic. General physical examination had shown no congenital defect. On examination of the chest it had been noticed that the two sides were symmetrical, but that respiratory excursions were almost completely absent on the left side. Percussion and auscultation showed the heart to be displaced into the right chest. Its action was rapid, but the sounds were clear and regular. Breath sounds were heard everywhere over the right chest except over the area of cardiac dullness, but over the left side they were heard only over the apex of the lung, both anteriorly and posteriorly. Over the rest of the left chest gurgling sounds were audible.

These findings induced her physician to have fluoroscopic examination and x-ray pictures made of the chest on the eighth day. They showed the heart to be in the right thorax, the esophagus angulated at the cardia, and the stomach in the left thorax (Fig. 1).

As time went on the condition became worse, there seemed to be slight relief during feeding when the baby was turned on her right side, but on the whole there was no change except that it lost weight continually during the first four weeks. The suffering of the child was distressing to the mother and it was therefore sent to the hospital in the hope of finding relief. The birth weight had been 6 pounds, and on admission four weeks later it was 6 pounds, 5 ounces. During the period of observation at the hospital the outstanding feature was cyanosis associated with every attempt at feeding. Each time an ounce of milk had been taken the baby became blue, had great respiratory distress, and then would become quiet and seemed on the point of dying. With the use of oxygen it would gradually recover and after a while more food could be given. There was no vomiting. Attempts were made to administer food in different positions but that did not improve the symptoms. With the most careful attention to diet, and supplementing it by hypodermoclyses the weight increase was but four ounces in two weeks.

Meanwhile the symptoms became worse rather than better and it was distressing to see the baby during a feeding period. Several x-ray studies were made, largely for the purpose of determining which organs had slipped into the left thorax, and partly to determine whether we were dealing with a hernia of the diaphragm or an

*Read at the Fourteenth Annual Meeting of the American Association for Thoracic Surgery at San Francisco, July 1, 2, 3, 1931.

eventration. These showed that soon after a meal the stomach was apparently rotated, with the greater curvature upward (Fig. 3). This rotation with possible angulation at the pylorus was held partly responsible for retention of the meal beyond the six hour period (Fig. 4). The pictures also revealed that a portion of colon was situated in the hernia and that there was undue delay in its emptying (Fig. 5). This was later verified with a barium clysma.

Our roentgenologist, Doctor William H. Stewart, contended that we were dealing with an eventration and advanced in favor of this diagnosis the presence of what he believed to be a high diaphragm, indicated by a curved convex line on the roentgen films (Figs. 1 and 2). Several of our surgical colleagues who saw the

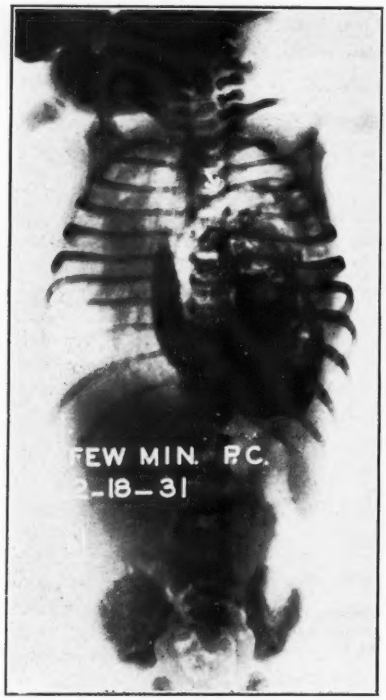

Fig. 1.

Fig. 2.

Fig. 1.—Indicating large dilated esophagus; angulation at cardia; stomach in left chest; heart in right chest.

Fig. 2.—Before test meal. Heart in right chest. Arrows point to diaphragm.

patient and the plates were in favor of the diagnosis of hernia on the theory that only a hernia could produce such severe symptoms and be responsible for retention of bismuth in the stomach for more than six hours (Fig. 4).

The question of etiology was considered. The onset of symptoms soon after a difficult labor with forceps extraction might speak in favor of injury to the phrenic nerve and subsequent paralysis of the diaphragm. There was nothing else, however, to support such a belief, for there was no evidence of associated injury in the neck or arms. The same history of early onset might as well be advanced in favor of the diagnosis of congenital hernia.

The advisability of operation was discussed repeatedly. It was felt that if we were dealing with an eventration the condition might adjust itself and conservatism would be in order. Considering it a hernia on the other hand, containing both stomach and colon, and associated with severe symptoms and retention of bismuth in the stomach and colon, would call for operation. Meanwhile the patient did not gain, the cyanosis increased and was so severe that oxygen had to be given almost continuously.

Consultation with the parents resulted in the decision to do an early operation. They wished to save the child further suffering and have it cured if that could be done.

Fig. 3.

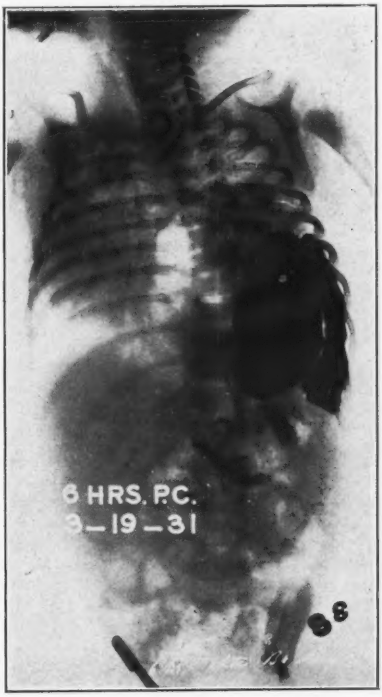

Fig. 4.

Fig. 3.—Test meal twenty minutes after ingestion, showing rotated stomach, with greater curvature upward.

Fig. 4.—Showing six hour gastric residue. Heart on right side. Diaphragm outlined.

Under general anesthesia an incision was made parallel to the left costal margin, beginning close to the ensiform cartilage and extending outward for about four inches. Small intestines presented. Exploration with a finger revealed a large defect in the left diaphragm. After the opening was brought into view a portion of the colon, the spleen and the entire stomach could gradually be drawn out of the chest. The colon and spleen were reduced quite easily but the stomach was drawn out only with considerable difficulty. It was distended, quite thick walled, and there was considerable gurgling while pulling it out. The organs were not adherent to each other and had apparently not been constricted abnormally. As soon as released

they would slip back into the thorax. It required considerable force to place these organs into the abdomen because the abdominal tension was considerably increased by their presence. Small pads and tampons had to be used to hold them there. After thoroughly exposing the diaphragmatic opening it was found that it was irregularly round in shape. It had thick, rounded, smooth, apparently muscular margins, and it looked as if the peritoneum were reflected from the under surface of the diaphragm over it into the thorax. There was a definite margin anteriorly, posteriorly, and laterally, but internally none could be seen or felt. At this point the tip of the left lobe of the liver projected into the thorax. Palpation of the thoracic cavity with the finger showed it to be empty. The heart could be made out on the right side, but no lung was felt. All the ribs were plainly palpable from within (Fig. 6).

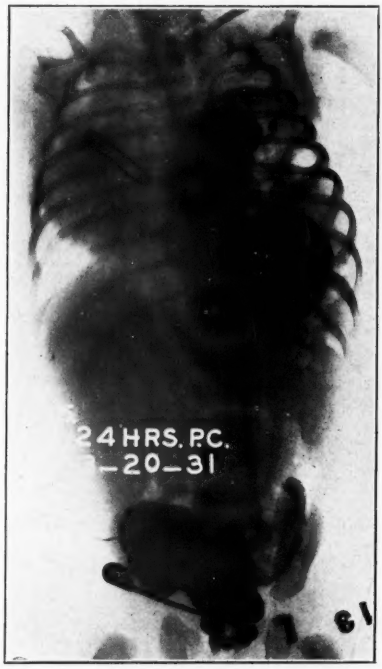

Fig 5.—Twenty-four hour retention in colon within the "hernial" sac.

Repeated attempts were made to draw the tip of the liver out of the thorax but it could not be reduced, it would always slip back. There was danger of tearing it. Forceful pull on it brought into view a whitish structure which was interpreted to be the esophagus.

We were apparently dealing with a diaphragmatic hernia with an open hiatus on the inner side. It was therefore decided to leave the tip of the liver in the chest and to close the hernial opening up to the liver. By grasping the outer margin of the hernial opening with an Allis clamp and making traction on it, the round opening was converted into an elongated one. Without much difficulty and without undue tension the opening was then closed up to the liver by means of three silk sutures and a continuous chromic catgut suture (Fig. 7). After placing the abdominal organs into normal position, the abdomen was closed without drainage.

The child had stood the operation quite well. It regained consciousness, and seemed bright. It was seen several times during the evening and nothing unusual was noted, except that the heart apparently remained in the right side of the thorax. A little breast milk and water were given without vomiting, and additional fluid was given by hypodermoclysis. Oxygen was administered through a nasal catheter.

The patient died unexpectedly about fourteen hours after operation. No definite cause could be stated. Death was attributed to "shock."

Autopsy permission was not granted, but a wound inspection was done which showed the abdominal wound well united. The abdominal organs were found where they had been placed. The suture of the diaphragm was perfect. After removing these sutures and reexamining the pleural cavity some serosanguinous fluid was found to have accumulated there.

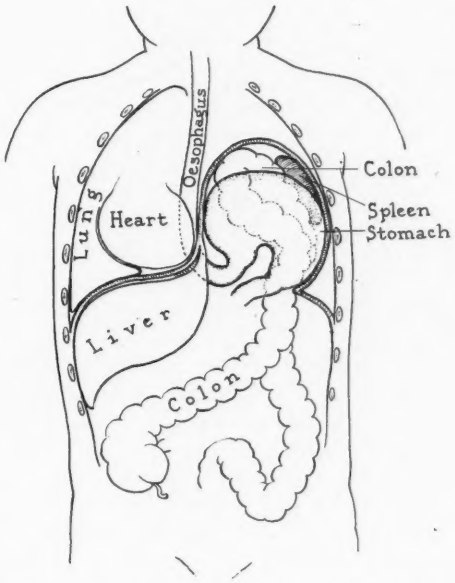

Fig. 6.—Diagrammatic sketch of conditions before operation, based on x-ray studies.

An attempt was made to determine why it had not been possible to draw the left lobe of the liver out of the thorax. Finger exploration of the chest showed no lung. The heart was apparently still on the right side. While pulling hard on the liver the same whitish structure came into view which during the operation had been interpreted to be the esophagus. Continuing the pull gradually brought a large sac out of the thorax which was eventually completely everted and found to be a large, stretched-out, central tendon of the diaphragm. It was so large that it had apparently almost filled the pleural cavity. It was so long that it could easily be brought out to the margins of the abdominal incision.

Our diagnosis therefore had to be changed from diaphragmatic hernia to eventration of the diaphragm, but that was not a satisfactory explanation. In the sense that the abdominal organs were still below the diaphragen the condition was an eventration, but in the sense that they actually had entered the thorax through a restricted aperture it was a hernia.

It is difficult to trace the embryologic development of the diaphragm, but it is necessary for the proper understanding of the various forms of abnormalities.

There are 3 main varieties of maldevelopment of the diaphragm:

1. A false congenital hernia is one without a sac. It is really a defect in the diaphragm due to failure of its various components to fuse properly. Abdominal contents may slip through this defect and lodge in the thorax without any covering.

2. A true congenital hernia on the other hand develops through a weak spot in an already formed diaphragm. As the abdominal contents

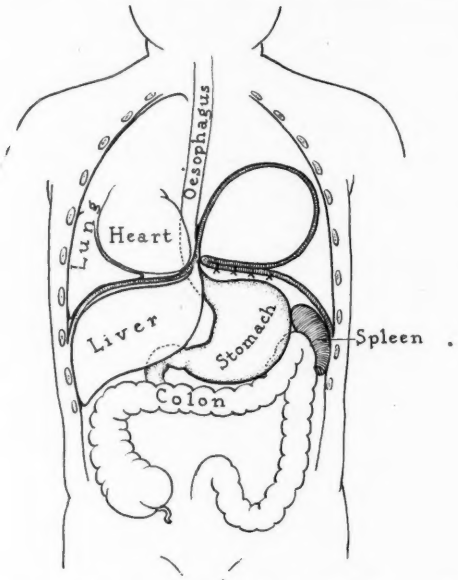

Fig. 7.—Diagrammatic sketch of conditions after operation, indicating reposition of organs and suture of diaphragm up to the lip of the left lobe of the liver which projected into the chest.

slip into the chest the peritoneum and pleural layers covering the weak spot are pushed ahead and form the sac of the hernia. This condition is much less common than false hernia.

3. Eventration of the diaphragm is comparatively rare. It may be congenital or acquired. There is no defect or hole in the diaphragm, it is simply relaxed and atrophic and bulges into the thorax often to the level of the third or fourth ribs. The congenital form is said to be due to failure of development of the muscle elements or of the phrenic nerve. Not much is really known about the underlying cause. The acquired type is occasionally seen as a complication of an acute infectious disease

probably by the toxin acting on the nerve. It may also follow trauma to the neck in which the phrenic nerve is injured, as for instance during difficult labor.

In either event one finds or expects to find a dome shaped bulge extending from the diaphragmatic attachments into the thorax. One would expect a uniform bulge, and one might reasonably think that the circumference at the base, meaning at the diaphragmatic attachments, would be larger than at the apex of the dome. In that case no constriction of the contents would occur.

Our case, although an eventration, does not quite correspond to such a picture. It was no doubt an eventration, but it seemed at the same time to be a hernia. The explanation seems to lie in the finding that apparently not all the elements of the diaphragm were involved in the process. It was the central tendon which had become stretched to form the large intrathoracic sac while the muscular elements were sufficiently developed to form a thick margin of what looked like a hernial ring. It appeared to be a localized eventration, or even a sacculated or diverticular eventration, due to partial paralysis or weakness of the diaphragm.

The treatment of a similar case would be surgical. Had we known the exact pathological condition we could have pulled out the sac the same as we did during the wound inspection and either plicated it, or it could have been attached with its dome to the abdominal incision. This would have been a very simple procedure and would have effectually separated the abdominal and thoracic cavities. Had the operation been done from above the sac could also have been plicated and sutured, or it could have been partly excised and the remaining material sutured by overlapping it. There was plenty of tissue for suturing and repair. As it was there was no difficulty approximating the muscular margins of the incomplete ring, only it left an empty sac above. Probably that would have made no difficulty. Any secretion which would form in there could be expelled through the opening close to the liver, and later the surfaces probably would have fused.

Had we not done a wound inspection the case would have been regarded by us as a false congenital hernia. We had a roentgenographic diagnosis of eventration, and we were prepared to find such a condition. We had hoped by means of a suction apparatus to pull out the sac in the same way that a diverticulum of the bladder can be pulled out. It was the appearance of a hernia with a thick hernial ring which misled us. The outcome probably would not have been different, however. It is conceivable that the additional manipulation might have resulted in a fatality on the table.

These patients with such an extensive congenital lesion may have associated changes in the vascular and respiratory systems which reduce their resistance a great deal. With eventration in general it would seem

wise to adopt a waiting policy in the hope that the condition may adjust itself. If the symptoms are severe, however, and one is dealing with a partial or sacculated eventration, operation is indicated.

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DISCUSSION OF PAPERS BY HARRINGTON AND EGGERS

DR. H. BRUNN.—I thought it might be interesting to report a series of congenital diaphragmatic hernias found in one family. The mother and father of this group are dead and we have no record of this condition in either of them.

The mother gave birth to ten children, two of whom died. We have been able to obtain x-rays of the eight remaining children, all of whom gave evidence of diaphragmatic hernia, none of which are very large but are definitely present. All of them enter the diaphragm through the esophogeal hiatus and all of them are symptomless.

I happened to discover the first hernia in a woman fifty-six years of age, who came to me with a cancer of the breast. An x-ray of the chest discovered a diaphragmatic hernia which had caused no symptoms. She told me that another sister, who was under the care of Dr. Herron, also had this condition. Dr. Herron then had x-rays taken of the rest of the family, who are now living in San Francisco, and in each one, a diaphragmatic hernia was found.

The patients' ages are fifty-six, sixty-six, sixty-three, sixty-four, sixty-three, fifty-four, fifty-two and fifty-two, the latter being twins, both males.

DR. F. A. C. SCRIMGER.—During wartime some of us had an opportunity to see a good many of the diaphragmatic hernias very shortly after injury. There were two things that seemed to be impressed on me at the time: first, the extraordinary respiratory distress; second, the absolute rigidity of the left side of the abdomen if the injury was on the left side. The relief that was obtained and the comfort found upon the repair of these hernias was also impressive.

I have had an opportunity of doing and seeing three traumatic hernias immediately, or almost immediately after injury. The diaphragm had been torn away from the chest wall, leaving a large rent. In none of them had we difficulty in repairing the rent from above the diaphragm.

I only had one opportunity of seeing the type of hernia through the esophageal opening and that was recently. And in that connection I would like to ask Dr. Harrington to be a little bit more specific in describing the method of repair—because in that case I failed to maintain the closure. The patient was a young boy of fourteen, running a very rapid pulse, having been vomiting a great deal. I got the structure down to the abdomen and repaired it, as I thought, satisfactorily; suturing I don't know exactly what tissues, but they seemed fairly stout tissues around the esophagus. Apparently from the symptoms the tissues held perfectly well for four days, and then there was a little pain and a little vomiting. And while he did not complain, probably because the opening had been enlarged, of anything

like the severity of the symptoms before, he did show on the x-ray that the hernia had recurred and therefore the maneuver had been unsuccessful.

If someone could give us a little information that is more definite on control, I would be very much pleased to hear it.

DR. C. D. LOCKWOOD.—I have had three cases of diaphragmatic hernia in civilian life and saw a number of cases as Dr. Scrimger did, during the war. Two of these cases I recognized in civilian life before operation. It seems to me that if we keep in mind the fact that this does occur, that there are certain very definite symptoms, and if we keep in mind these symptoms and signs, we can recognize these cases without the x-ray. Of course the x-ray should confirm the diagnosis.

There are four or five very definite clinical symptoms. First, the retraction of the abdomen, this was very characteristic in these two cases that I recognized. The second, the fixation of the costal margin. The third, the presence in the chest of abnormal sounds, such as gurgling and the absence of the respiratory murmur. The lung is compressed and you get practically no expiratory murmur. Then the absence of the normal movements of the diaphragm up and down upon the chest. The displacement of the heart to the right side of the chest is found in a majority of the cases of long standing. And if we are on the lookout we should recognize these.

There is one other point I want to emphasize in one case that I lost, and that is the importance of changes in intraabdominal pressures in these cases. This particular man came to California, not realizing he had any trouble whatever with his stomach or any other part of his body. En route he developed very acute appendicitis and vomited excessively. He was taken off the train and to the hospital. We operated for acute appendicitis. He developed symptoms of obstruction and we thought it was due to appendiceal adhesions. On reopening him I discovered the herniation through the diaphragm but was unable to release it and the man died of shock.

So it seems to me if we keep in mind the possibility of these things, especially in association with acute abdominal trouble, we may often recognize them even without the x-ray.

DR. S. HARRINGTON.—In response to Dr. Scrimger's question as to the treatment of the repair of the enlarged esophageal opening in the diaphragm, the repair depends upon the type of esophageal opening that is present. In my experience there have been practically three types of closure; first, the small opening in which the esophagus is close to the right side of the esophageal opening and is closed by placing a stomach tube through the esophagus and doing an overlapping of the margins of the opening up to the esophagus, the innermost suture incorporating the esophagus in the closure. The stomach tube in the esophagus at the time of closure prevents too tight closure of the opening around the esophagus and prevents obstruction of the esophagus. In some of the larger openings, the esophagus will be in the center of the enlarged esophageal opening. In many of these cases it is necessary to close the esophagus both from the right and left sides incorporating the esophagus in the opening. In a few instances, it is necessary to make a three-sided closure, the third side coming from below beneath the left margin of the liver causing a three-cornered closure of the esophagus. The amount of overlapping depends upon the amount of tissue present which can be utilized in the closure. In some of the larger openings there is considerable loss of structure and the overlapping is accordingly less.

In regard to the possibility of recurrences, I believe that the removal of the sac has as much if not more to do with the prevention of recurrence as closure of the opening, as it is the sac of the hernia which is the primary factor in dragging the stomach through the opening into the thoracic cavity. It is very important that the sac is either completely removed or completely disconnected from the stomach and hernial ring and allowed to remain in the thorax.

THE IMPORTANCE OF THE RESPIRATORY MOVEMENTS IN THE FORMATION AND ABSORPTION OF PLEURAL FLUIDS*

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ALTHOUGH collections of fluid in the pleural cavity are very commonly met with in both medical and surgical practice, and although much is spoken of "exudates" and "transudates," when one comes to examine the question more closely, it is seen that our knowledge of the factors influencing the formation of these effusions is actually rather meager.

In 1921 E. A. Graham,[1] in proposing an explanation for the rapid accumulation of large quantities of fluid in the pleural cavity in cases of acute streptococcal pneumonia, suggested the part played by the respiratory movements was of importance. Thus, he argued, given an edematous lung and edematous pleura, it seemed that the increased negative pressure of inspiration might actually *suck* fluid from the lung and pleura into the pleural cavity, much as fluid can be sucked from edematous tissues by a needle and syringe. In addition, given areas of lung which were unable to expand fully as a result of obstruction or occlusion from the inflammatory process there would be an even greater sucking force exerted on these parts when a deep inspiration was taken.

In order to prove the truth of this argument he constructed an artificial chest by connecting a suction pump to a bell-jar in which was suspended a pair of lungs, the trachea being fastened to a glass tube passing through a cork in the top of the jar. Lungs from a freshly killed dog were placed in the "chest" and an "edematous" condition obtained by pouring down the trachea water to which a few drops of acetic acid had been added. In other cases lungs affected with bronchopneumonia (either human or from dogs) were used. Under the influence of the respiratory movements a copious exudate was then obtained in the jar, but instead of the flow occurring during inspiration, it was seen to occur during expiration; as the lungs expanded no fluid appeared, but with expiration a stream of drops would run from them into the jar. If methylene blue was added to the fluid in the lungs it was seen to fill the subpleural lymphatics with the act of inspiration and then with expiration to appear through the pleura with the fluid.

It was argued from this that during the lowered pressure of inspiration fluid was sucked into the subpleural lymphatics, and that when expiration occurred, there was a sudden diminution in the area

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of the lung surface and the volume of these lymphatics, and in consequence some of the fluid contained in them passed through into the pleural space.

Although there appeared little doubt that such a sequence of events happens in the living body as well as in a dead preparation, it seemed that two important differences were introduced which needed justifying before a final proof could be arrived at. First, there is the question of altered permeability occurring in a dead as opposed to a live membrane; second, with the presence of a circulation in a living lung fluid might be absorbed and carried away which in a dead lung could not be accommodated in the intrapulmonary lymphatics and tissue spaces.

In order to investigate the conditions obtaining in the living body it was decided to carry out a similar set of experiments using a "heart-lung" preparation of the Starling type in which, however, the heart and lungs, after the establishment of the artificial circulation, were removed from the body and placed in a glass "chest" so that direct observations could be made of the passage of fluid through the pleura.

Numerous difficulties were encountered in setting up such a preparation but eventually a technique was perfected by means of which a satisfactory "set up" could be obtained in almost every instance. As full descriptions of the steps of the heart-lung preparation are not easily found in the literature it is proposed to give a careful account of the steps used in this work.

DESCRIPTION OF APPARATUS (FIG. 1)

The artificial chest used was a large specimen jar in the lid of which a hole had been cut big enough to take a No. 12 rubber cork; it is important to note that the hole was cut so that the cork could be inserted from the under or inner surface of the lid (Fig. 2). The bottom of the jar was divided into two compartments by a glass partition some two and one-half inches high, held in place by plasticine (*a*, Fig. 1). Through the cork passed five structures (Figs. 1 and 2):

(1) An arterial connection (*c*)
(2) A venous connection (*i*)
(3) A large cannula for the trachea
(4) A connection to a respiratory pump (*j*)
(5) A connection to a manometer

A special clamp (*b*, Fig. 1) was made to hold the lid firmly in place on the jar.

The requisite negative pressure for inflation of the lungs was obtained by exhausting air through a T-piece (not shown in the picture) on the tube *j* and the normal respiratory variations of pressure were then obtained by means of a Starling's respiratory pump which removed and replaced an appropriate volume of air as often as necessary per minute;

it was connected to the jar by the tube *j*. The arrangement of the rest of the apparatus (i.e., the artificial circulation) is as recommended by Starling,[2] and needs only brief description.

The arterial blood leaves the heart by means of the tube *c* which has a T-tube connection with a mercury-manometer which records arterial

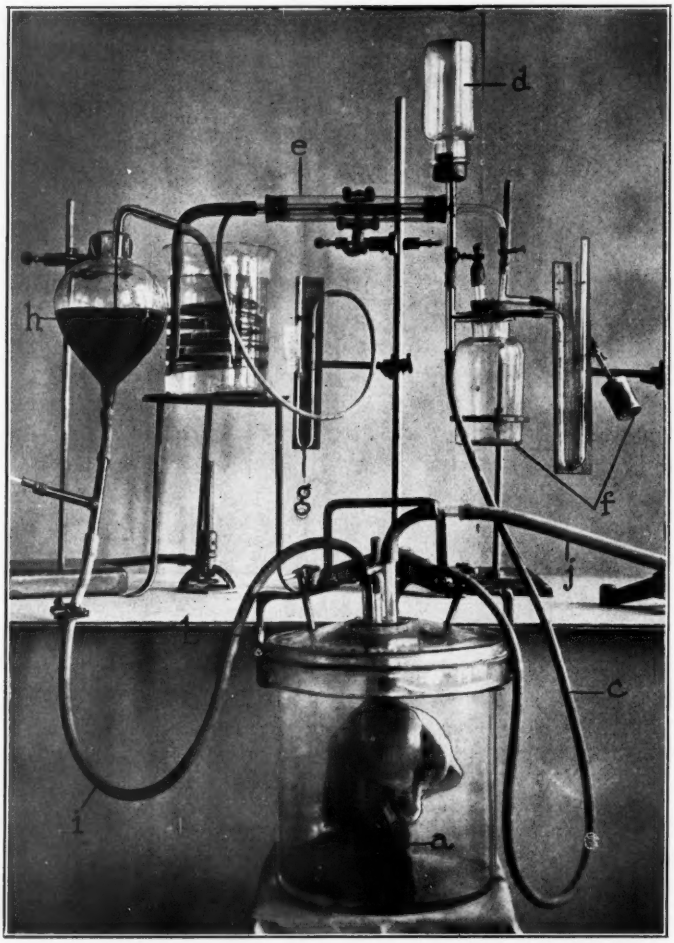

Fig. 1.—A photograph of the apparatus and preparation. It is to be noted that for the sake of clarity the artificial chest has been placed below the bench level; during the experiment it is always on the bench itself.

(*a*) Partition dividing bottom of jar into right and left halves; an effusion is present in the right side.

(*b*) Special clamp to secure lid. (*c*) Arterial tubing in which is included a T-piece attachment to an Hg manometer. (*d*) Buffer bottle. (*e*) Resistance consisting of a rubber finger cot surrounded by an air-containing tube.

(*f*) Hard pump and pressure bottle by means of which resistance can be regulated. (*g*) Hg manometer recording resistance. (*h*) Venous reservoir. (*i*) Venous tubing in which is a T-piece carrying a thermometer. (*j*) Tube leading from respiratory pump to jar.

pressure; *d* is an ordinary bottle whose air content serves as a buffer replacing the normal elasticity of the vessel walls; the normal peripheral arterial resistance is represented by the tube *e* which surrounds a rubber finger cot through which the blood has to pass. The air pressure in this tube can be raised and controlled by means of the pressure bottle and hand-pump *f;* a manometer *g* measures the amount of this pressure that is exerted on the outside of the rubber cot; this pressure is the resistance which the blood has to encounter on reaching *e*. After meeting this resistance the ordinary intermittent output of the heart is transformed into a steady stream which passes through the coil in the water-bath where the temperature is regulated and then into the venous reservoir *h*. A tube *i* (into which is let a T-piece carrying a thermometer) then conveys the blood back to the right auricle. In all these experiments a constant venous pressure was maintained by keeping the reservoir at the same height, the inflow from the reservoir being regulated by a screw-clamp. It is to be noted that although in the photograph for the sake of clarity the artificial chest has been momentarily dropped below the level of the bench, during the experiment it is on the bench level.

Before commencing the experiment it was found advisable to have at least 500 cubic centimeters of defibrinated blood (procured from another animal) in the venous reservoir *h*. It is possible to use Ringer's solution in the reservoir and then supplement it with the animal's own whipped blood, but it is not so satisfactory.

The animal is anesthetized with ether and tracheotomy is immediately performed. The tracheotomy cannula is then connected to a positive pressure pump preparatory to opening the thorax. The midline tracheotomy incision is then extended as far as the xiphisternum and the chest rapidly opened by dividing the sternum in the midline. If this is done boldly and the midline is not transgressed, the two halves can be widely separated, and by clamping the internal mammary vessels on each side at once, almost no blood need be lost. Occasionally it is necessary in addition to catch some of the lower intercostal vessels. The chest should then be held widely open by a self-retaining retractor.

The first step now is ligation of the brachiocephalic artery, and this vessel can be easily and quickly exposed by wiping apart the two layers of the anterior mediastinum with a gauze-covered finger. The ligature is passed and tied immediately above the point of division and, since now the blood supply to the brain has been cut off, the administration of ether can be stopped. The left subclavian artery is ligated and a long ligature then passed round the arch of the aorta immediately beyond the origin of the subclavian but is as yet left untied. A similar loose ligature is placed round the inferior vena cava. Continuing in a clockwise direction the vena azygos is ligated in two places and divided and then the superior vena cava is tied and a cannula inserted below the ligature.

This cannula is previously filled with defibrinated blood in order to prevent an air lock. The dissection is completed by inserting a cannula in the brachiocephalic artery; it is important that this cannula should be as large as possible as we feel this is a factor in the prevention of early cardiac failure and resultant pulmonary edema, which can be a too frequent occurrence in these preparations. The connection from the venous reservoir to the venous cannula is now filled with blood and passed over the cannula in the superior vena cava. Similarly the arterial cannula is connected with the arterial tubing.

It will be seen that the arterial system has a T-tube at its commencement (Fig. 2); this useful addition to the apparatus was recommended to us by Dr. I. de Burgh Daly to whom we owe our thanks. Care must

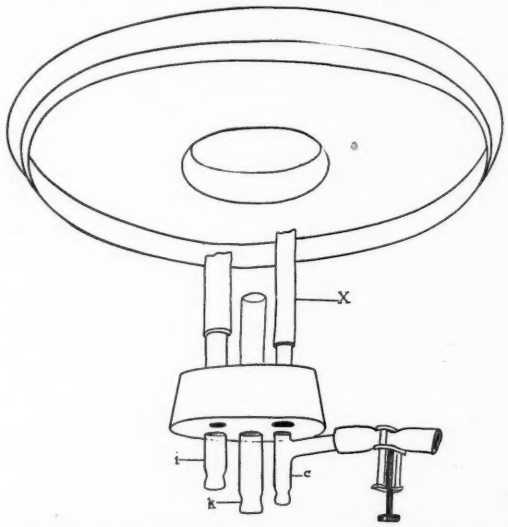

Fig. 2.—This is to show the retrograde manner in which the cork fits into the lid and to illustrate in more detail the connections in this region. Two holes in the cork (one for the respiratory pump and one for a manometer) have been left empty in order to display more clearly:

(*c*) The arterial connection. (*k*) The tracheal connection. (*i*) The venous connection.

It is to be noted that (*i*) and (*c*) are not cannulas but are attached to the cannulas by short rubber connections which allow of some freedom of movement. (*X*) denotes the point at which the tube is clamped while the animal is being bled through the T-piece.

be taken to see that the arterial tube is clamped off above the T-piece (at the point indicated in Fig. 2), otherwise clotting will occur in the system. The clamp on the T-piece is now opened and the aortic ligature tightened; blood then spurts out of the side-limb and can be collected in a beaker and defibrinated; when the flow begins to fall off (the hind part of the body should be first raised in order to aid the flow) the flow from the venous reservoir to the superior vena cava is turned on and the ligature on the inferior vena cava tightened. Sufficient defibrinated

blood is run through the heart and lungs to wash out all coagulable blood, the clamp on the arterial tubing is then opened and the limb of the T-piece closed. Blood now rushes round the artificial circuit into the reservoir and back to the heart and lungs once more.

The trachea is next cut across and tied to the tracheotomy tube passing through the cork; this is hidden in the photograph of the apparatus but is shown clearly in Fig. 2. The heart and lungs can now be rapidly and easily removed from the body by dividing the necessary structures; great care must be taken at this stage not to inflict lacerations on the lung tissue else leaks will occur which will be likely to ruin the whole experiment.

The cork is now fitted in a retrograde manner into the lid of the chest, the preparation placed in the jar so that right and left lungs occupy the respective halves of the jar, the top is clamped down and the positive pressure apparatus exchanged for the respiratory pump. The lungs now empty and fill in a manner identical with that occurring in the body, air is drawn in and out of the trachea in the normal manner, and the rate and depth of respiration can be easily regulated. The circulation is well maintained and such a preparation can be kept going perfectly satisfactorily for at least two hours and probably for as long as four hours; it has not been found necessary in these experiments to prolong the time much longer than two hours. On many occasions after two hours satisfactory working the preparation has been broken up and the heart has continued to beat for many minutes although cast aside and receiving no blood.

The use of a partition in the bottom of the jar enables any fluid to be collected in the appropriate half of the chest; as can be seen in the photograph, an effusion is present covering the bottom of the right half of the jar.

It was decided that in order to prove or disprove the occurrence in the living lung of the mechanism suggested by Graham it would be necessary to introduce perfectly standard conditions in two sets of experiments. First a pair of dead lungs was taken and into the right main bronchus was inserted an obturator in the form of a rubber cork with a glass tube passing through it; a rubber connection passed out through the trachea. When the dead preparation had been working for a few minutes fluid (from 75 to 100 c.c., according to the size of the dog, of Ringer's solution to which ⅓ of 1 per cent acetic acid and a few drops of methylene blue had been added) was introduced into the lower right lobe by means of the tube and obturator. In this way it was proved by repeated experiments that an effusion would begin to appear in the corresponding side of the jar within five to fifteen minutes of the introduction of the fluid; this fluid would be stained by the dye and would also be slightly blood-stained. The same relation of the outpouring of fluid to expiration could be easily verified as described by Graham; further-

more, the part played by the respiratory movements in the formation of the effusion could be demonstrated by showing that cessation of the respiratory movements resulted in cessation of the flow of fluid.

EXAMPLE:

Experiment 6.—

12:20 P.M. 100 c.c. of fluid introduced via tube and obturator into right lower lobe.

12:35 P.M. Definite unilateral effusion present. From this time on fluid increased steadily until

12:55 P.M. when experiment was stopped; 9 c.c. of fluid were present in the right side of the jar. The preparation was left in the jar but motionless until

1:35 P.M. (next 30 minutes) when no formation of fluid occurred.

On recommencing respiratory movements, fluid again began to collect.

The same procedure was applied to a heart-lung preparation and it was proved conclusively that a unilateral effusion could be produced and showed the same relation to expiration and inspiration in its formation.

EXAMPLE:

Experiment 11.—

Weight of animal—8 kilos

Dissection started—10:30 A.M.

Artificial circulation established—11:00 A.M.

Completed in jar—11:10 A.M.

The heart was maintaining a good pressure and a good output; the preparation was quite dry, no oozing at all taking place. At the end of five minutes there was no sign of any pleural fluid forming and at

11:15 A.M. 85 c.c. of fluid (Ringer, plus 1/8 of 1 per cent acetic acid) were slowly poured into the right lower bronchus by means of a previously introduced obturator.*

11:30 A.M. A definite thin, blood-tinged effusion was just commencing to appear in the right side of the jar, none appearing on the left side. As the flow increased the relation to expiration could be easily demonstrated; *no dye was seen to be present in the fluid in the jar.*

There was a steady increase of fluid on the right side until 12:15 P.M., and up to this time *none on the left;* however, a rather early pulmonary edema now began to supervene and some bloody fluid began to appear on the left and at the same time the fluid on the right became more blood-stained. The fluid was removed from the right chest and the experiment terminated. Thirty-five cubic centimeters had been collected in one hour. Examination revealed the obturator firmly in place in the right lower bronchus. No dye could be demonstrated in the fluid.

The objection may well be raised that pouring fluid into a bronchus does not give rise to conditions exactly comparable to a true pulmonary

*The amount of fluid that should be introduced into the trachea was worked out on a basis of 20 c.c. per kilo of body weight, this being the maximum amount that Winternitz[3] has shown can be accommodated in the lungs without overflow; usually one-quarter of this full amount was used.

edema. Evidence has been obtained to answer this objection from two further sets of experiments.

PULMONARY EDEMA DUE TO LEFT-SIDED HEART FAILURE

Everybody who has worked with the heart-lung preparation mentions only too feelingly the liability to the onset of a severe pulmonary edema after the preparation has been going for a shorter or a longer time. We found that in certain cases when the artificial circulation was opened up there would be for some obscure reason a temporary failure on the part of the heart to maintain a good output; after a short time the strength and vigor of the heart would improve, but in such cases an early onset of a rapidly progressive pulmonary edema almost always followed. Shortly after the preparation was placed in the "chest" fluid would begin to appear from the pleural surfaces of both lungs and rapidly accumulate in both sides of the chest. In these cases the flow on expiration was very easily demonstrated indeed.

EXAMPLES:

Experiment 4.—

Dissection began—2:40 P.M.

Circulation established—3:20 P.M.

There was a temporary heart failure when the artificial circuit was opened, but the heart soon picked up and maintained a good output. On removing the viscera from the chest, however, it was clear that an early bilateral pulmonary edema was present. No fluid appeared until the lungs had been in the artificial chest for some fifteen minutes; at this time a frothy blood-stained fluid began to form in the trachea and simultaneously a blood-stained effusion began to collect in each chest; the experiment was stopped at 4:15 P.M. owing to rapid depletion of the contents of the venous reservoir.

Experiment 8.—

Dissection began—2:45 P.M.

Artificial circulation established—3:05 P.M.

The heart was dilated and beating feebly; only a low pressure was registered. Massage was started and at 3:15 P.M. a good circulation was being maintained. By this time, however, there was a generalized pulmonary edema of moderate degree and when the setup in the jar was completed at 3:45 P.M. fluid began to appear at once. It was deeply blood-stained and a similar frothy fluid appeared in the trachea. At 4:25 P.M. the heart began to show signs of weakening and the experiment was accordingly stopped. There were 110 c.c. of fluid in each chest; i.e., 220 c.c. had accumulated in forty minutes.

The relation of the flow to the act of expiration was clearly demonstrable. As the degree of edema increased, the rate of effusion increased, until at the end the formation was so rapid that the venous reservoir had to be constantly refilled owing to depletion of the volume of circulating fluid. It is quite clear from these and many other similar experiments that with a pulmonary edema of cardiac origin rapid outpouring of fluid can occur into the pleural cavity, and that this out-

pouring bears a definite relationship to the respiratory movements of the lungs.

INFLAMMATORY EDEMA PRODUCED BY IRRITANT GAS

Considerable thought was expended in an endeavor to procure a controlled unilateral condition of edema strictly comparable to the inflammatory edema that is present in a true pneumonic process. It was finally decided that such a condition could best be produced by the use of an irritant gas such as chlorine; the large amount of experimental work conducted during the war[4] has shown quite clearly that exposure to these gases results in the early onset of an acute inflammatory edema which progresses to a definite pneumonic process after some twenty-four hours.

Phosgene apparently produces a more rapid and intense edema than chlorine,[4] but owing to greater ease and safety in manipulation chlorine was used.

The method employed was as follows: The animal would be anesthetized with sodium amytal given intravenously, and by means of bronchoscopic manipulation a similar obturator to the one used in the previous set of experiments was passed into the right main bronchus. The tube passing through the cork was now connected to a flask containing chlorine (usually 1:500 or less) and by means of a rubber balloon attached to the flask thorough ventilation of the right lower lobe with the gaseous mixture was carried out for five minutes. This was done in the afternoon and the following morning a heart-lung preparation was set up.

EXAMPLE:

Experiment 20.—

Weight of animal—8½ kilos.

April 30: at 12 noon the right lower lobe was gassed for five minutes with a chlorine mixture by means of a tube and obturator in the right lower bronchus.

May 1: Animal awake and lively; no dyspnea; occasional cough; some patches of frothy mucus in cage.

Dissection commenced—10:30 A.M.

(A few c.c. of free fluid were present in the right pleural cavity and the right lower lobe was seen to be irregularly gassed.)

Dissection completed—11:05 A.M.

In jar —11:20 A.M.

After five minutes a thin slightly blood-stained fluid began to appear on the right hand side; the areas of gassing in the lower lobe were not uniform and the affected areas stood out as semitransparent edematous patches and from these fluid could be seen to drip with every act of expiration. The heart was beating very satisfactorily; the left lung showed no signs of any gassing or edema; it was quite dry.

At 12:20 P.M. the fluid in the right chest was collected and found to measure 14 c.c. This fluid was allowed to stand overnight and the red cells settled as a small sediment at the bottom (less than 1/10) the overlying fluid being

clear. An Esbach determination showed the amount of albumin present was 2¼ parts per 1,000.

Examination of the specimen showed a typical gassing confined to the right lower lobe; the rest of the lung was normal. The lobe was heavy, exuded plenty of fluid on section and was almost uniformly gassed in the central part, the peripheral parts being more patchily affected; areas of emphysema alternating with areas of acute edema. The solid areas sank in water. The pericardium contained a slight excess of blood-stained fluid.

It was thus shown that in living lungs with a well-maintained circulation, it is possible to demonstrate the part played by the respiratory

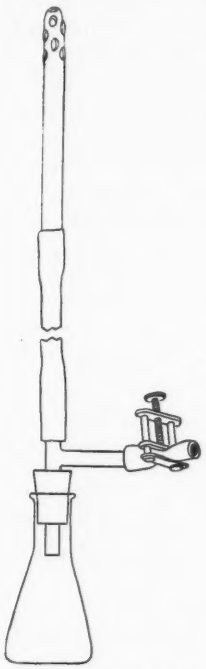

Fig. 3.—Illustrates the many holed, large bore glass cannula used for draining the pleura. A purse-string through the skin and muscle layers fastens it securely in place. Fluid collects in the flask, and the T-piece can be used to take a manometric reading or can be connected to a tambour to obtain a record of the respiratory movements.

movements in the formation of a pleural effusion when an edema exists either of a vascular or an acute inflammatory nature.

It is, however, possible to raise various objections to such a heart-lung preparation in that numerous uncontrolled factors are introduced which may not play a part in the living body itself. The object of using the artificial chest was actually to observe the relation of the formation of a pleural effusion to the acts of inspiration and expiration. It remained, therefore, to demonstrate first that under conditions similar to

those obtaining in the heart-lung experiment an effusion could be shown to appear in the intact animal, and second to demonstrate more fully the part played by the respiratory movements in the rate of formation of the fluid; a step which was difficult to achieve in the heart-lung work.

Accordingly, an experiment similar to the introduction of acidified Ringer's solution into the lung was first attempted.

An animal was anesthetized with sodium amytal given intravenously, and by means of a bronchoscope an obturator was introduced into the right lower lobe bronchus as in the chlorination experiments; the fluid (Ringer's solution plus ⅓ of 1 per cent acetic acid and a few drops of methylene blue) could now be introduced into the right lower lobe in a well-controlled manner. Before fluid was poured in, a cannula was introduced into each pleural cavity and connected to a small flask as shown in Fig. 3. A T-piece allowed manometric readings to be taken when necessary; a large cannula with several holes was used so as to render occlusion very unlikely. These cannulas were made to lie parallel to the lung surface and in the lower posterior part of the chest; a purse-string through the superficial tissues secured them in place.

EXAMPLE:

Experiment 12.—

Weight of animal—12 kilos.

An obturator was introduced into the right lower bronchus and a cannula placed in each pleural cavity. Their presence in the pleura was verified by taking a manometer reading; this was always repeated during the experiment.

At 4:05 P.M. fluid was introduced into the right lower lobe and by 4:20 P.M. 100 c.c. were in. An occasional drop of blood-stained fluid had flowed from each cannula, doubtless owing to the certain inevitable trauma of introduction. The amount on each side was, however, negligible; i.e., a few spattered drops on the right and less than 1 c.c. on the left in thirty-seven minutes. At 4:30 P.M. both flasks were removed and cleaned; from 4:32 P.M. onward fluid was collected.

On the left side an occasional drop of deeply blood-stained fluid appeared. On the right side a steady stream of drops appeared and fluid rapidly accumulated in the flask; it was clear and only faintly tinged with red cells. Respiratory movements were moderate, but never powerful. At 5:12 P.M. (forty minutes) the flow had virtually ceased and the experiment was terminated.

Period of collection—40 minutes.
Fluid from right side—12 c.c.
Fluid from left side—2 c.c.

The animal was then bled through the carotid and the chest opened in the midline without contamination with blood. Both cannulas were in the pleural space and both were patent. The one on the left had caused a little trauma to the adjacent pleura which had doubtless resulted in the 2 c.c. of bloody fluid on the left; about ½ to 1 c.c. of blood-stained fluid was in the left chest. The right cannula had caused no demonstrable damage; about 2 c.c. of blood-tinged fluid were in the right pleural cavity. The left lung was normal, the obturator was securely in place in the right bronchus and the right lower

lobe alone showed edema and local staining with dye. No dye was observed in the pleural exudate, either in this experiment in which methylene blue was used or in similar ones in which trypan blue was used.

Esbach on fluid from pleura: 2 parts of albumin per 1000.

Similar experiments were conducted with a gassed right lower lobe. An obturator would be passed the night before and the lung gassed as

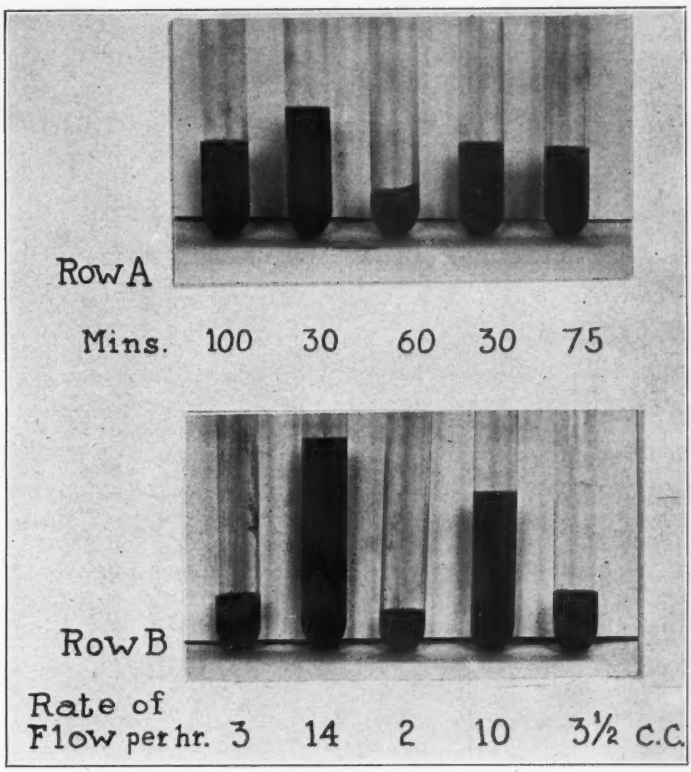

Fig. 4.—Shows variations in the rate of formation of pleural fluid depending on type of respiration. The upper row of tubes (*A*) is a photograph of the fluid collected in Exp. 14; the time intervals are shown underneath. The lower row (*B*) has been made up to illustrate more simply the variations in the hourly rate of flow in each of the upper tubes. The fluid has been darkened in order to make it show up well.

in the earlier experiments; on the following day cannulas were placed in each pleural cavity and fluid was shown to be forming on the affected side.

An experiment conducted in this way was then used to demonstrate the effects of variations of the respiratory movements on the formation of pleural fluid as shown by the rate of flow from the cannula.

EXAMPLE:

Experiment 14.—

Weight of animal—13½ kilos.

April 22. 3:40 P.M. Obturator passed into right main bronchus and lung ventilated for five minutes with a chlorine mixture of approximately 1:250; this was performed under sodium amytal anesthesia.

April 23. 9:00 A.M. Animal awake and alert but showed signs of respiratory disturbance; respirations rapid and somewhat harsh. Anesthesia was again induced with sodium amytal and at 11:15 A.M. cannulas were inserted into each pleural cavity and their position verified by taking a pressure reading. In a short time fluid (clear with slight tinge of blood) began to appear from the right side and in ten to fifteen minutes some 2 to 3 c.c. were present. The flow then became slower and every effort was made to empty the chest of any fluid by (a) gentle pressure, (b) occluding the trachea momentarily and thus causing the lungs to distend. In this way one was reasonably sure that any accumulated fluid was drained off; it seemed that these first two or three cubic centimeters were doubtless a small collection in the chest before the experiment started.

The flow continued very slowly indeed, only an occasional drop appearing; the respirations were very quiet.

12:30 P.M. CO_2 (30 per cent) was administered by means of a catheter passed through a tracheotomy tube.

12:35 P.M. With the response of the respirations to CO_2 fluid now began to appear and so the flask was emptied and replaced. The contents measured 5 c.c.

i.e., 5 c.c. in 100 minutes.

The administration of CO_2 was continued for thirty minutes and at 1:05 P.M. the flask was again emptied. There had been an immediate increase of flow and a steady drop, drop of fluid from the cannula; 7 c.c. were in the flask.

i.e., 7 c.c. 30 minutes.

During this time respirations were deep and forceful but not rapid. The administration of CO_2 was now stopped and a further small amount of sodium amytal (0.1 gm.) given to depress respiration.

1:05-1:35 P.M. Respirations slow and shallow although quite regular; as after thirty minutes there was scarcely 1 c.c. of fluid, collection was continued for a further thirty minutes.

2:05 P.M. Flask emptied and contained 2 c.c.

i.e., 2 c.c. in 60 minutes.

2:10 P.M. Once more ventilated with CO_2 and flow at once started briskly and steadily.

2:40 P.M. Flask emptied and contained 5 c.c.

i.e., 5 c.c. in 30 minutes.

Quiet respiration was again permitted, although the movements were beginning to be slightly more vigorous than before.

3:55 P.M. Experiment terminated; flask contained 4½ c.c.

i.e., 4½ c.c. in 75 minutes.

At no time during the experiment had any appreciable amount of fluid appeared on the left side; a manometric reading showed the left side cannula was patent and in the pleural cavity.

A postmortem was now performed and both cannulas were seen to be patent and in the pleural space; neither had inflicted any visible trauma.

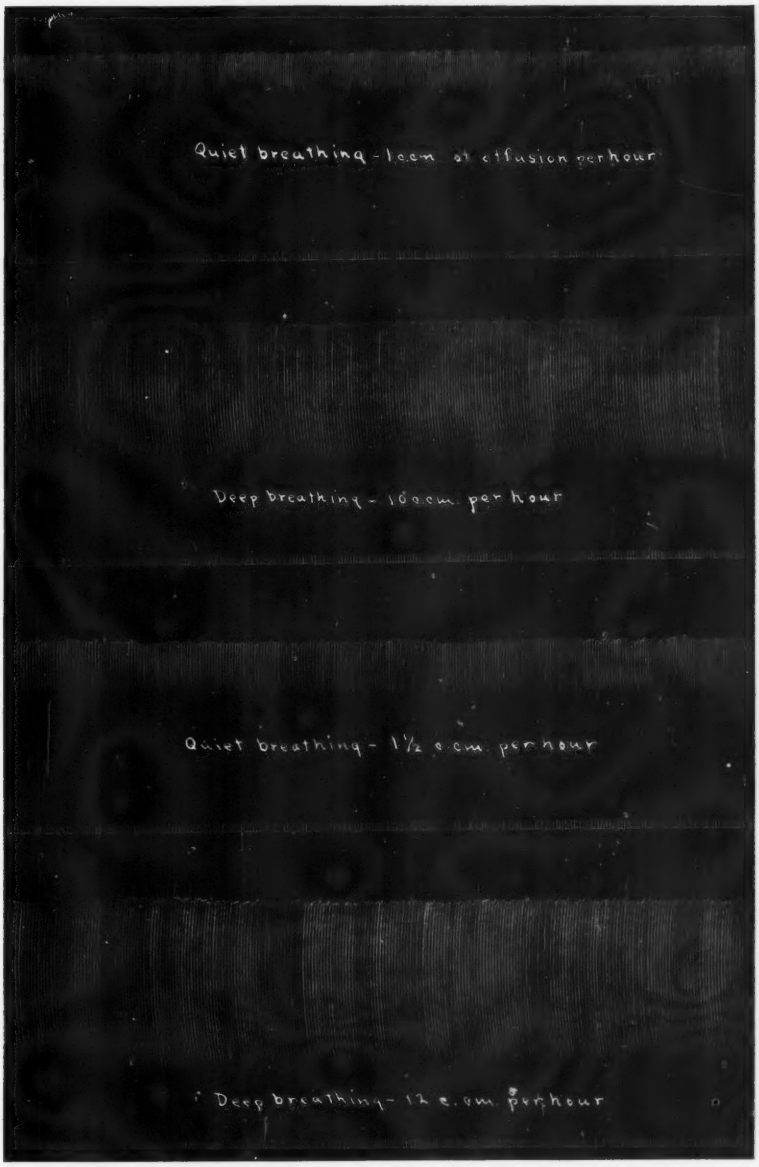
Quiet breathing - 1 c.cm of effusion per hour
Deep breathing - 16 c.cm. per hour
Quiet breathing - 1½ c.cm. per hour
Deep breathing - 12 c.cm. per hour

Fig. 5.—This is a drum-tracing of the respiration in an experiment similar to the one depicted in Fig. 4, the animal being gassed the night before and cannulas introduced the next day. The record was obtained by a connection to a cannula in the pleural cavity; it is, therefore, a pressure record. In this case the time intervals were equal (thirty minutes) and the increased flow with increase of respiration and the diminished flow with shallower respiration is very marked.

Left pleural cavity—contained less than 1 c.c. of fluid.
Right pleural cavity—contained less than 2 c.c. of fluid.
The right lower lobe showed a typical gassed appearance and was almost solid with edema; it sank at once when placed in water; its macroscopic and microscopic features were identical with those recorded by Winternitz and others in cases of gassing with chlorine. The rest of the lungs was normal.

Row A of Fig. 4 is a photograph of the tubes containing the fluid collected over the indicated periods; the rate of flow per hour is made concrete by the tubes of row B which clearly show the variations in the rate of formation of the pleural fluid with the variations in the respiratory movements. The increased flow with deeper and the diminished flow with shallower and feebler respiration are well marked. The fluid has been darkened for purposes of illustration.

Fig. 5 is the record from a similar case in which kymographic tracings were taken by connecting the T-piece on one of the cannula connections to a tambour. The weight of this animal was 10 kilos and autopsy revealed a well-gassed lower right lobe. The tracings show very well how increased depth of respiration (obtained by partially occluding the trachea and administering CO_2) results in a greater formation of fluid and a diminished respiratory excursion with a smaller pressure change gives rise to a much smaller amount of fluid.

It is, of course, not suggested that these amounts represent all the fluid exuded from the lung; absorption is going on simultaneously and a certain amount of fluid is returned to the circulation in this way without being drained off by the cannula.

In these experiments performed with a cannula it can, of course, be argued that the trauma and friction of the cannula may be responsible for the increased flow of fluid; a control cannula has been used in the opposite side of the chest, but it can be argued that friction of this cannula against a normal lung might not produce fluid whereas it might with an edematous lung. Accordingly, an experiment was planned to test this point.

EXAMPLE:

Experiment 26.—
Weight of animal—11:75 kilos.
After anesthetization with sodium amytal an obturator was passed into each lower bronchus and thus both lower lobes were comparable. In the right chest was inserted a solid glass rod of the same size as the cannulas used in the previous experiments. Between 11:30 A.M. and 11:45 A.M., 60 c.c. of acidified Ringer's solution were introduced into each lower lobe by means of the tube and obturator. The animal was given alternating fifteen-minute periods of quiet and forceful respiration. At 12:45 P.M. the experiment terminated. From previous experiments it was known that fluid should collect in the pleura; if the friction of the glass cannula was responsible for a greater production of fluid from the corresponding lung, then more fluid should be present in the right side of the chest; since absorption was also occurring during the experiment it was not expected to find any large amounts of fluid.

An autopsy was performed and the solid "cannula" was seen to be satisfactorily situated in the right pleural space; both obturators were in place and both lower lobes showed equal "edema." From the right pleural space 1½ c.c. of fluid were removed by suction and from the left side 2 c.c. Far from being an excess on the side of the cannula, there was actually a slightly smaller amount.

This experiment seems to demonstrate quite clearly that the presence of the cannula does not of itself affect the flow of pleural fluid. Similarly experiments have been conducted with symmetrical treatment of the lungs and a patent cannula in one side; they showed that the amount of fluid obtained through the cannula is greater than the amount removed from the opposite pleural cavity at the end of the experiment only by as much as might easily have been absorbed during the period of the experiment.

Since Ringer's solution with a few drops of acetic acid in it had been shown to pass readily through the pleura, it was thought necessary to investigate the behavior with plain Ringer's solution. Accordingly, Ringer's solution was introduced into the lung on one side in the same way and both pleural cavities controlled with cannulas. We were able to show that a pleural fluid was also formed every time that Ringer's solution was used, although the amount collected was never so much as with acidified Ringer's solution or a gassed lung.

EXAMPLE:

Experiment 22.—

Weight of animal—13 kilos.

125 c.c. of Ringer's solution introduced into the right lower lobe; between 11:15 A.M. and 12:30 P.M. 6 c.c. of fluid were obtained from the cannula.

i.e., 6 c.c. in 1 hour 15 minutes

It will be seen from later remarks that such an amount can be readily dealt with by the normal rate of absorption of fluid from the pleural space, and in bilateral experiments of this nature we were indeed unable to demonstrate any *accumulation* of fluid in the chest at autopsy on the side on which a cannula had not been used. In other words, the presence of a cannula would result in fluid being drained off before it could be absorbed.

Since it is easy to show that a simple solution such as this passes readily through the pleura, one is led to speculate as to the importance of this phenomenon as a mode of entry of infection into the body. For instance, in view of the preceding observations and of the work on the absorption of particulate matter from the pleural cavity,[5] showing that carmine or India ink particles pass readily from pleura to lung or from mediastinum to pleura, it seemed not unlikely that organisms might take this same path. In order, therefore, to test this possibility, we introduced into the lung Ringer's solution to which had been added a few cubic centimeters of a dead broth culture of streptococci; we were able to show that the organisms were recoverable almost immediately from the pleural space.

EXAMPLE:

Experiment 33.—

Weight of animal—14¼ kilos.

An obturator was passed into the right lower bronchus in the usual way and a cannula introduced into the right pleural space. Into the right lower lobe was slowly poured 100 c.c. of Ringer's solution to which had been added 3 c.c. of a killed broth culture of a hemolytic streptococcus. Within a minute or two of the introduction of the solution, fluid began to drip from the cannula in the pleural cavity (previously none had appeared in twenty minutes). A first sample was collected after five minutes and a second sample then collected over the next half hour. Both of these specimens of fluid from the pleural cavity were shown to contain large numbers of streptococci, some singly, some in pairs, and others in short chains. None were seen lying intracellularly.

An autopsy was performed and the position of the cannula found to be satisfactory; the obturator was firmly in place and the lower right lobe alone contained the solution. The left pleural cavity, however, contained 2 c.c. of quite clear fluid and this also was found to contain a few organisms, though not nearly so many as the fluid on the right had contained. The passage of fluid across the mediastinum is well recognized and occurred uniformly in some later experiments of our own; it is very likely that the fluid in the left pleural cavity collected in this way; if such is the case it is interesting in that the organisms were able to pass through two sets of membrane although far fewer were present on the left side. It is realized that this portion of the work has not been fully worked out and much remains to be done concerning it. It was, however, thought necessary to include it since it elaborates still further the earlier work.

Quite clearly infection of the pleura can occur in this way and without necessarily a simultaneous parenchymatous lesion of the lungs; although one would naturally expect the latter to occur also, as indeed it commonly does. Apart from giving rise to a local lesion, however, we have to consider the possibility of this being a mode of entry of a blood-stream infection and it may well be that some of the cases of septicemia with no obvious primary focus have, indeed, commenced in this way. The contention that has always raged around the mode of onset of an acute lobar pneumonia can also be considered in this light. Thus, there is some doubt as to whether the condition is primarily a septicemic process which rapidly localizes in the lung or whether the lung condition is primary and the septicemia which is frequently present in the early stages secondary to it. In the earliest stage of a pneumonia the affected area is markedly edematous, and in the light of our experimental work it would be very unlikely if fluid rich in organisms did not pass into the pleural space and there be absorbed with an early production of a septicemia. The almost constant early pleurisy can also be explained in this way, especially in those cases in which there is no certain evidence of a direct involvement from a parenchymatous spread.

ABSORPTION FROM THE PLEURAL SPACE

From a consideration of the conditions which have been shown in the above work to influence the formation of excessive collections of fluid

in the pleural cavity, it does not seem at all unlikely that a similar mechanism may be acting under normal circumstances. That is, with each respiratory movement a small amount of fluid is pumped into the pleural space and the normal moist condition of the membranes maintained. At the same time it is apparent that if such a steady flow of fluid, although of small amount, is taking place into the pleural cavity, there must be a well-regulated absorption occurring simultaneously; otherwise there would be a progressive filling of the chest with fluid. Moreover with excessive respiratory movements (such as occur in prolonged exercise) larger formation of fluid should occur* and there should be a greater tendency for a collection to form under such circumstances unless the rate of absorption was also capable of responding to or dealing with the increased formation. It seemed, therefore, on a priori grounds probable that the rate of absorption from the pleural space might also be affected by the character of the respiratory movements.

Absorption of fluids from the pleural cavity has been investigated by Starling and Tubby[6] and Starling and Leathes[7] and they were able to demonstrate, using an isotonic salt solution, that absorption occurs from the pleura at a steady rate; they found that the average rate of removal of such a fluid was 5 c.c. per hour. They, however, make no mention at all of the type of respiration exhibited by the animals with which they worked and whether there was any variability in this important mechanism. A search of the literature, moreover, reveals no experimental work on the question of a relation between the rate of absorption from the pleural space and the type of respiration.

Several workers have, however, pointed out the part played by the respiratory movements in the flow of lymph from the abdomen to the chest; Ludwig and Schweigger-Seidel in 1866 were able to demonstrate that the respiratory movements alone could cause substances to flow through the lymphatics; they eviscerated a dead rabbit, hung it head down and placed a colored gelatin solution in the concavity of the diaphragm, and after performing artificial respiration were able to show that the lymphatics of the diaphragm were filled with the solution. In 1903 McCallum[8] performed a similar experiment and showed that particulate matter could pass into the lymphatic system of the diaphragm from the peritoneal cavity and thence into the chest when all other factors (such as circulation and cell-activity) had been excluded and respiratory movements alone played any part.

Bolton,[9] in 1921, discussed the same question and was able to show that a suspension of solid particles (carmine or india ink) placed in the

*Some evidence of such a mechanism has been obtained. Thus, in one experiment in which an attempt had been made the previous night to gas the right lower lobe it was found that when cannulas were introduced into the chest only a small flow of fluid occurred and this was unusually clear, scarcely showing even a tinge of blood; with quiet respiration the flow was at the rate of 1 c.c. per half hour, and with forced respiration 2 c.c. per half hour. At autopsy it was found that owing to some error of technic no gassing of the lung had occurred. It is felt that in this case a sample of the normal output of pleural fluid was obtained.

subphrenic space was absorbed and was demonstrable in the sternal glands in a much shorter time if the respiratory movements were made more violent.

Again, in 1922, Wadsworth[10] discussing absorption from the pleural space suggested that the respiratory movements acted as a pump and forced fluid into the lymphatics and thus were responsible for the normal absorption of fluid from the space. He did some experiments to prove this and demonstrated that a suspension of carmine or india ink placed in the pleural cavity was soon found in the lymphatic spaces of the intercostal and visceral pleura; by performing artificial respiration

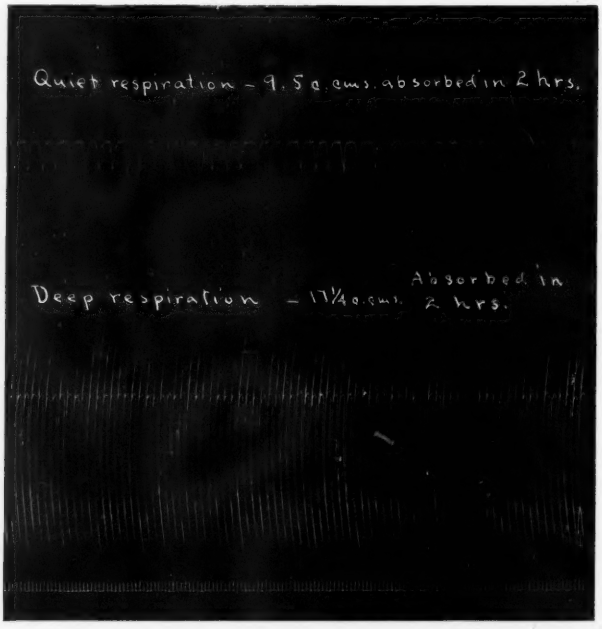

Fig. 6.—Respiratory tracings from two animals in which 50 c.c. of Ringer's solution were introduced into the pleural cavity. The first animal with quiet respiration absorbed 9½ c.c. in two hours, and the second animal absorbed 17¼ c.c. in two hours with deep respiration.

on dead animals he was able to show that a similar absorption took place and assumed that the respiratory movements thus normally played a part in the circulation of fluid in the thoracic cavity.

Dybkowsky,[11] in 1866, suggested a mechanism of absorption into the lymphatics which was dependent upon the respiratory movements. Thus "with each inspiration the intercostal subpleural lymphatic network is opened out and filled with fluid from the pleural cavity, the forces affecting this being the elasticity of the lung on one side and the tightening of the intercostal fascia by the inspiratory movements on the other.

With each expiration the intercostal fascia is relaxed and bulges inward, the capacity of the subpleural lymphatic network is diminished and its contents are emptied into the efferent lymphatic vessels. This pumping action varies with the vigor of the inspiratory movements and the elastic force exerted by the lung."

(It is to be noted that in this paper no attempt is made to discuss the relative parts played by the blood vessels and lymphatics in the absorption of fluid from the pleura.)

In no case, however, is there any attempt to show a relation between the rate of absorption and the character of the respiratory move-

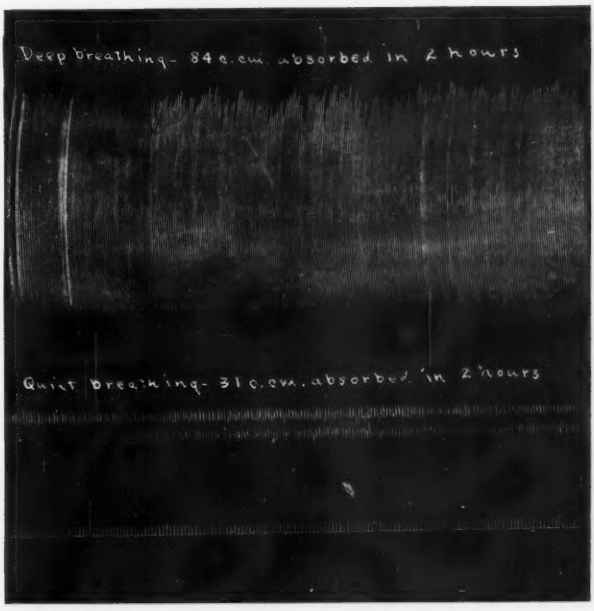

Fig. 7.—Respiratory tracings of two animals in which 200 c.c. of Ringer's solution were introduced into the pleural cavity. The first animal after two hours of deep breathing absorbed 84 c.c.; the second animal after two hours of quiet breathing absorbed only 31 c.c.

ments; attention seems to have been wholly concentrated on the fate of particulate matter introduced into the body spaces. It was decided, therefore, to conduct some experiments to determine whether the same factors which influenced the passage of fluid into the pleural cavity also influence the rate of its removal.

Two dogs were taken of approximately the same weight and after anesthetization with sodium amytal, 50 c.c. of Ringer's solution at body temperature were introduced into the right pleural cavity of each. One of the dogs was arranged to breathe quietly (larger dose of amytal and a short wide cannula in the trachea) and the other made to breathe

more forcefully (by partially occluding the tracheal cannula); a cannula was introduced into the left chest and connected with a tambour and a record of the respiratory movements taken on a revolving drum. At the end of two hours an overdose of sodium amytal was injected into the carotid, the chest opened in the midline and the remaining fluid carefully removed by suction.

It is to be noted that in all these experiments in spite of the fact that the fluid was introduced into one pleural cavity it was found at autopsy to be in both cavities with no demonstrable passage between the two pleural cavities.

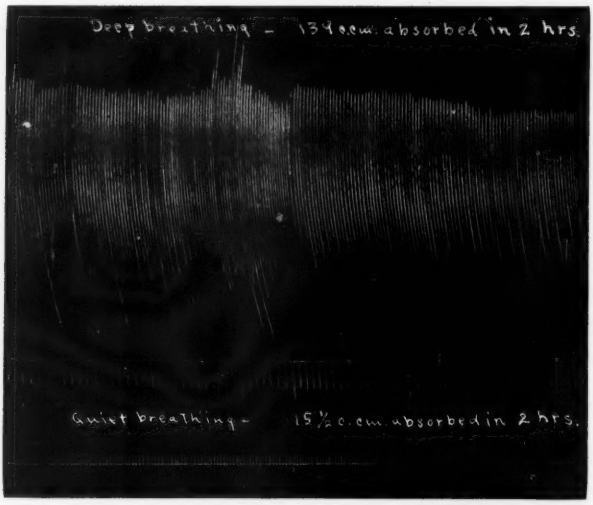

Fig. 8.—A similar tracing to that shown in Fig. 7. In this case the first animal absorbed 139 c.c. in two hours and the second animal absorbed 15½ c.c. in the same time. In each case 200 c.c. of fluid were introduced.

EXAMPLE:

Experiment 25.—

In the case of the animal with quiet respiration,

9 c.c. were absorbed in two hours.

In the case of the animal with forceful respiration,

17.25 c.c. were absorbed in two hours.

The weight of the first dog was 6.35 kilos, and of the second 4.75 kilos. Thus, although the second was a smaller animal it showed with deeper respiration an absorption rate of very nearly 100 per cent more (Fig. 6).

Since it can be argued that with the deeper respiration there is more agitation of fluid and consequently more surface contact over which absorption may occur, other experiments were conducted using much larger amounts of fluid. Thus, if it was a question of surface area alone there should be an approximation between the rates of absorption with quiet and forced respiration.

Two animals were taken and similarly treated, but with 200 c.c. of fluid in the chest instead of 50 c.c. One was made to breathe quietly and one forcefully. A kymographic tracing of their movements is shown in Fig. 7. After two hours the animal with quiet respirations was found to have absorbed 31 c.c.; and the animal with deep respirations had absorbed 84 c.c. The weights of the animals were 4.8 and 5 kilos respectively.

A similar experiment showed absorption rates as follows (Fig. 8):

With quiet respiration—15½ c.c. absorbed in 2 hours.
With forceful respiration—139 c.c. absorbed in 2 hours.
Weight of animals—4.8 and 5.6 kilos respectively.

It is thus apparent that although with the larger amounts (200 c.c.), the general rate of absorption is faster than with the smaller amounts (50 c.c.) of fluid, showing that surface area plays some part in absorption, there can be no doubt at all that the rate of absorption is profoundly influenced by the force of the respiratory movements. For instance, each of these animals, although quite small, would have completely removed 200 c.c. of fluid from its chest in less than four hours when exhibiting hyperpnea.

The presence of such a mechanism is very easily understandable and could almost have been predicted. It means that the deep respirations produced by a large accumulation of fluid in the chest almost certainly are effective in hastening the removal of that fluid.

Of course, under pathologic conditions of the pleura two other conditions have to be considered: (1) the simultaneous more rapid formation of fluid as shown earlier in this paper, and (2) the possibility of an impaired power of absorption on the part of the pleura. Starling[7] showed that the absorptive power could in no way be affected either by scalding the lining with hot water, or by treatment with sodium fluoride; on the other hand, Corfer[12] has obtained evidence that in the presence of an acute pleurisy absorption is delayed. It is, however, suggestive that in a patient with an early streptococcal effusion the rate of absorption of toxic material from the thorax may well be much greater than one would anticipate. The rapid accumulation of fluid to form the large effusions simply means that the rate of formation is far in excess of the rate of absorption but gives no indication of the degree of absorption that is indeed taking place; this can be merely a matter for speculation at the moment.

Again, in those experiments with the heart-lung preparation in which a severe degree of pulmonary edema supervened, there was a very rapid accumulation of fluid in both sides of the artificial chest. In the similar condition of acute pulmonary edema as it occurs in man a collection of pleural fluid is not usually noticed at autopsy. It is to be remembered, however, that in this condition there is also severe hyperpnea on account of the filling of the air passages with abundant frothy fluid, and it is highly probable, therefore, that the pleura is capable of absorbing large quantities of fluid at a correspondingly rapid rate.

Furthermore, in these cases the pleura is quite healthy and the question of impaired power of absorption does not arise. It is not surprising, then, that no gross excess of fluid is found in the pleural cavity although from our work with the heart-lung preparation, it is almost inconceivable that a free flow from lung to pluera is not taking place.

It is realized that the experiments on this variation in the rate of absorption are incomplete, but it was thought opportune to make mention of the evidence in this article.

SUMMARY AND CONCLUSIONS

1. An adaptation of the heart-lung preparation of Starling as modified by Daly is described, by means of which the exudate from each lung may be separately collected for a period of hours.

2. In this way it has been possible to show that in a living preparation the formation of a pleural effusion is dependent upon the respiratory movements, a finding which corroborates the conclusions previously drawn by Graham from experiments which utilized dead lungs in an artificial thorax.

3. Under direct visualization fluid can be seen to drip from an edematous lung in expiration, and it is suggested that the rise in negative pressure during the act of inspiration fills the subpleural lymphatics, and when expiration follows, these filled lymphatics empty part of their contents into the pleural space.

4. An edematous condition has been produced, (a) by pouring acidified fluid directly into the bronchi, (b) by means of passive congestion, and (c) by an active inflammatory process, namely, exposure to a dilute chlorine mixture.

5. On the living intact animal, after an edematous lung has been produced either by methods (a) or (c), fluid can be collected by means of a cannula inserted into the pleural space.

6. In this way it has been possible to show that the rate of formation of the fluid is dependent upon the force of the respiratory movements and the associated pressure changes. A quiet, easy respiration produces a small effusion; a deep, labored breathing causes a much more rapid accumulation.

7. In addition to fluid of an inflammatory nature passing from the lung to the pleural space, it has been shown that an ordinary Ringer's solution, if introduced into the bronchi and alveoli, will, by the respiratory movements, be made to pass into the pleural cavity.

8. Part of a broth culture of a streptococcus was added to Ringer's solution and introduced into the lung in the same way. The organisms were found to pass through the pleura with the fluid almost immediately and without the aid of cellular activity. It is suggested that this may be an important mode of infection in the body and may indeed be the point of entry of some of those septicemias of obscure origin.

9. Just as the rate of formation of fluid is influenced by the character of the respiratory movements, so also is the rate of absorption of fluid affected. With quiet, easy respiration the absorption is slow; with deep labored breathing it is much more rapid. A large accumulation of fluid with a consequent large surface area of contact is in any case absorbed at a slightly more rapid rate than a smaller collection; this increase is, however, in no way comparable with the marked effect of increase of the force of the respiratory movements.

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DISCUSSION

DR. C. S. BECK.—I shall look with interest for the complete report of Dr. Brock's work. The problem that he has studied is of such fundamental importance that I am surprised that there is so little information on the subject. The rapidity with which a transudate is absorbed from the pleural space is probably the reason that it is so difficult to produce fluid in the chest experimentally by ligating the pulmonary veins or the venae cavae. Charles Bolton, in 1902, produced hydrothorax in cats by ligation of the superior vena cava. Karl Schlaepfer did not find hydrothorax in the dog after ligation of the pulmonary veins. I have been unable to produce hydrothorax in the dog by partial ligation of the pulmonary veins or by ligation of the superior vena cava. It may be that studies on the anastomosis between the bronchial veins and the pulmonary veins such as are being conducted by Dr. Reichert will be of assistance in determining whether hydrothorax is primarily due to failure of the right side of the heart or failure of the left side of the heart.

I wonder whether it is not preferable to conduct the experiments in the intact animal. It seems to me that any animal membrane will allow the escape of fluid by filtration in response to a negative pressure.

DR. F. REICHERT.—I don't know of any other place in the body where we can have the opportunity to study pure lymph and vascular edema that we have in the lobe of the lung, and that is what led us into this experimental work where after exposing the vessels of a lobe of the lung we deliberately attempted to destroy the lymphatics. I think you saw that illustration yesterday where we had a very small amount of clear watery fluid plus a tremendous fibrinous exudate that covered the whole lobe following lymphatic blockade to a lobe.

Securing that, we were interested in seeing whether we could produce, by interfering with the venous return from the lobe of the lung, a pure vascular transudate. In some previous unpublished work, where we put metal bands on the large veins of the body, on the inferior vena cava in the abdomen, in the thorax, and the superior vena cava in the thorax, we found in a number of experiments that in four or five instances a pleural watery exudate occurred. This led Dr. Johnson to attempt to produce the same type of reaction by blocking or at least partially occluding the veins from the lobe. And in doing that we found a number of interesting things. Repeated aspirations failed in many instances to secure any fluid, nor was there evidence of it on the x-ray. In one of several instances in which Dr. Mathes blocked the venous return from the lobe, there was a marked effusion without any fibrous exudate at all.

I still feel that the problem is by no means solved, and that perhaps we will work along different lines. Particularly this nice work of Dr. Brock's, that you presented, and the work that Dr. Beck is doing will perhaps lead to a solution of the problem.

IMPORTANT REFLEX RELATIONSHIPS BETWEEN THE LUNGS AND OTHER VISCERA*

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IN THE past it frequently has been said that "the most capable physician is the one who knows his pathology best," meaning his structural pathology. This statement holds true today, but the interpretation is much broader and includes not only structural pathology, but functional pathology as well.

Structural pathology, logically, should have been developed before functional pathology just as it was. It is more simple and lends itself to more accurate investigation. Then, too, the facts established in its investigation are useful and necessary to the development of the more difficult study of disturbances in function. While it was logical for the study of pathologic physiology to have followed that of pathologic anatomy, it was not logical for it to have followed it at so great a distance; for these subjects are not divorced but closely united and interdependent. Pathologic anatomy has meaning in disease largely as it is interpreted in terms of function.

Numerous outstanding salutary effects have come from the illogical division of medicine into specialties of this and that organ; of which one is the intensive study of the organs, themselves; another, the proof of the illogical nature of such a division of medicine. No sooner did men begin to study each organ diligently than they saw that it had no independent existence but was a part of the body as a whole, to influence and to be influenced by, other tissues and organs. As an outgrowth of this conception has come the study of pathologic physiology which attempts to discover and account for the changes which may be produced throughout the body as a result of disease or dysfunction of a given organ and also to find out in what manner a given organ responds to pathologic changes elsewhere in the body.

This is the essence of the understanding of clinical medicine. This is the natural superstructure to arise on the foundation of pathologic anatomy. It carries the student into the fascinating and inseparable studies of biophysics, biochemistry, and physiology. Endocrinology and neural physiology are of special importance because of their function of correlating and integrating the activity of all of the structures and organs of the body. My discussion today will be confined largely to the neurophysiology of the lung, considering the possible reflex changes in its function which may be caused by stimuli arising in other organs;

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and, on the other hand, the reflex changes in function of other organs which may be produced by stimuli arising in the lung. The physiologic mechanism which gives us pathologic function during disease is the same that provides normal physiologic function in health.

PULMONARY NERVES

The lungs are innervated by sympathetic fibers which arise mainly from cells in the stellate ganglia, the connector neurons joining the cells to the central nervous system having their cell bodies in the lateral ganglia of the upper five or six thoracic segments.[1] Stimuli arising in the lung may be carried back to these upper thoracic segments of the cord over the sympathetic afferent system, to be transferred to efferent neurons in the production of reflex effects. Instead of forming their somatic reflexes regularly as other organs, that is, in the same segment that receives the afferent stimulus, they form their somatic reflexes through the midcervical nerves. Their visceral reflexes, however, are produced through the upper thoracic sympathetic nerves. These efferent neurons of the sympathetic system which originate in the lateral horn of the upper five or six thoracic segments are subject to reflex effects which are produced by impulses carried to these segments by either somatic or visceral afferent neurons.

The parasympathetic fibers which supply the lung belong to the vagus nerve and arise in the medulla. The vagus mediates reflexes with the various cranial nerves, including other vagal afferent neurons, and according to Rasmussen[2] and Larsell,[3, 4, 5, 6] possibly in the midcervical nerves.

The action of the sympathetics and the vagus within the lung is antagonistic. The innervation of the lung is shown in Fig. 1.

Action of Nerves on Lung.—The vagus efferent fibers have the function of activating the muscles and glands of the bronchi; and, as a rule, of inhibiting inspiratory effort, making inspiration short and shallow. On the other hand, stimulation of the central end of the divided vagus may now and then increase the respiratory rate. The explanation of these opposing effects is that there are two sets of afferent vagus fibers—one inspiratory, the other expiratory—and that the expiratory effect (inspiratory-inhibiting effect) is the one which predominates. This double effect is met elsewhere, for example: in the cardiac sphincter where both closure and opening of the orifice are controlled by the vagus in response to stimuli which course centralward over afferent vagal fibers, and in the pupil which contracts and dilates in response to stimuli which are carried centralward over the optic nerve.

Respiration may be inhibited through impulses carried over afferent neurons other than vagal, as for example, over the Vth cranial nerve, as is frequently observed when irritating substances enter the nose; over the IXth cranial nerve during the act of swallowing, which pre-

vents foreign particles from entering the air passages, and again over the superior laryngeal branch of the vagus which, in supplying sensation to the larynx, provides a second protecting mechanism for the air passages and inhibits inspiration in case abnormal substances should succeed in entering the larynx.

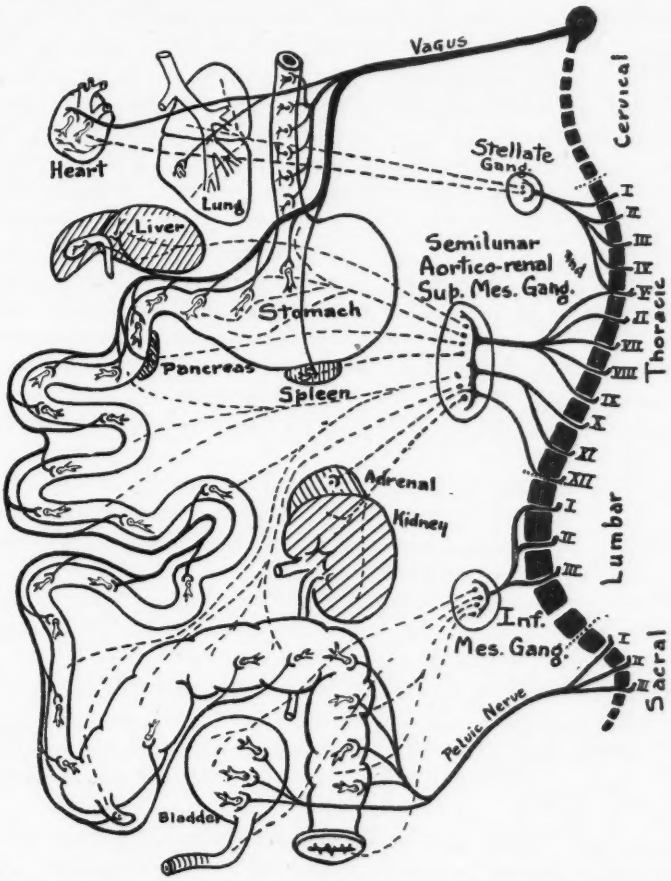


Fig. 1.—Showing the lung as a part of the enteral system, with which it has a common innervation. *1.* The sympathetics (broken lines) which supply the lung originate in the upper five or six thoracic segments, and pass to the lung as nonmedullated fibers from the stellate ganglion. *2.* The parasympathetics (solid lines) supplying the lung belong to the vagus and arise in the medulla. (From Pottenger: *Symptoms of Visceral Disease,* The C. V. Mosby Co.)

Respiratory effect may be increased, on the other hand, through emotion, the exciting impulse coming from without through the nerves of special sense, or through sensation carried to the central nervous system over spinal sensory nerves, or through emotions which originate within the individual.

There is some evidence to the effect that vagus stimulation dilates the pulmonary vessels; but this is not definitely established. The sympathetics inhibit both muscle and glands and cause vasoconstriction.

The effect of vagal stimulation is to shorten and diminish the lumen of the bronchi, reducing the lung volume, while that of sympathetic stimulation is to dilate and lengthen them, increasing the lung volume. In paroxysms of asthma, while the bronchi are constricted and shortened, the lung volume does not decrease, but, on the contrary, increases because of the interference with the exit of air from the lungs, and the resultant emphysema. Compared with massive collapse, asthma causes severe dyspnea because it is general throughout the lung instead of being confined to a lobule or lobe. The marked dyspnea, too, is accompanied by an excess of CO_2, which keeps the respiratory center stimulated.

In case of asthma the muscles of the constricted bronchi may be relaxed by measures which cause the sympathetics to overcome the vagal action, such as adrenalin, toxemia or severe fright, and by measures which inhibit the vagus, such as atropin. In massive collapse the vagal inhibition may be overcome by stimulating the respiratory center by CO_2 either with or without removal of obstructing secretion. Adrenalin and atropin may also be of value.

Action of Stimulation of Efferent Nerves on Bronchi.—The bronchi through their musculature have the function of constriction and dilation—of lengthening and shortening; and through their glands, of secreting a mucus for the purpose of moistening the surfaces. The bronchi also have a peristaltic rhythm which in all probability has a protective rôle similar to the cilia of the cells of the mucous membrane in keeping the air passages free from unusual substances.

Any reflex which expresses itself through the pulmonary efferent nerves may interfere with one or more of these functions. It may either constrict or dilate the bronchi, shorten or lengthen them, change the normal peristaltic rhythm or increase or decrease the secretion of the mucous glands.

Constriction and shortening of the bronchi and increase in the secretion of the mucous glands are recognized as parasympathetic effects which may be produced by activation of the pulmonary branches of the vagus. Dilatation and lengthening of the bronchi and decrease in secretion of the mucous glands are recognized as effects which may be produced by stimulation of the sympathetic or inhibition of the parasympathetic (vagal) nerves.

The lungs, too, have the function of carrying on respiration, an act which is controlled primarily by the CO_2 of the blood, but which may also be stimulated and inhibited reflexly through afferent impulses over the vegetative nerves.

PULMONARY REFLEXES

Two Sources of Pulmonary Reflexes.—A system of nerves consists of both afferent and efferent neurons, which serve to activate the tissues supplied and at the same time to pick up and carry centralward stimuli which arise in those tissues. Afferent neurons are essential to the participation of any given organ or tissue in the complex activities of the body as a whole.

The afferent fibers which belong to the sympathetic system have not lent themselves to ready study. Larsell[3, 4, 5, 6] practically ignores them in his study of the afferent neurons from the lung. In so important and so active a nervous system as that of the sympathetics in the lung, however, it would seem improbable that there should be afferent fibers, or that their action should be inconsequential to the lung. Then, too, afferent fibers belonging to the sympathetic system are recognized in all other important viscera, a function of which is to bring the nerves arising in the corresponding spinal segments into reflex activity with the viscus. The lung has reflexes corresponding to those produced by the sympathetic afferent system in other organs, and it seems but natural that it should have corresponding afferent nerves to initiate them. In this connection it must be understood that when we speak of the sympathetic afferent system we do not mean to indicate that these neurons are necessarily true sympathetic fibers. On the contrary, there is much evidence to show that the visceral afferent nerves belong to the skeletal afferent system.

Head[7] states that the visceral afferent nerves belong to the deep and protopathic systems of the body and are of the same order as those which supply the skeletal structures, and further, that the system which enables man to experience acute pain and make fine sensory distinctions in the skeletal structures are absent from visceral tissues.

The presence of these afferent neurons in sympathetically innervated tissues has been proved in most organs, and it would seem probable that they are present in all.

The efferent neurons of the parasympathetics arise in the nucleus solitarius of the vagus and pass out to the bronchial tissues.

Action Caused by Stimulation of Afferent Pulmonary System.—Stimulation of the afferent systems of neurons in the lung may reflexly alter the various activities of the lung itself, and also the activities of other organs and tissues. The stimuli which arise during normal function of a normal organ are not of such a nature as to excite pathologic reflex action. But unusual stimuli arising in an inflamed organ may reflexly disturb function in many organs; in fact, the possibilities are only limited by the number of efferent neurons with which the afferent neuron may mediate. I have suggested the paths for and described about forty reflexes which may be caused by stimuli arising in the lung.[1, 8, 9]

Peculiarity of Pulmonary Somatic Reflexes.—The developmental segmental relationship in the central nervous system is apparently the determining factor in locating the level of transference of afferent impulses to effector neurons in the production of reflexes. By knowing the segments in which the sympathetic connector neurons for a given organ arise we may know the spinal nerves which are most likely to complete the reflexes from that organ, according to Sherrington's[9] law "that each afferent neuron finds in the same segment of the cord that it enters an efferent neuron with which it mediates most readily in the formation of reflexes."

The important viscera seem to be regular in their reflex relationships except the lungs and possibly the thyroid gland. The lungs receive their sympathetic innervation from the upper five or six thoracic segments but express their principal skeletal reflexes through the IIId, IVth and Vth cervical spinal nerves. So, in order to account for this departure from the law we assume that the impulse, on entering the cord, is transferred upward through intersegmental neurons to the midcervical segments. The path of the thyroid sympathetic reflex seems to be the same as that of the lung.

I have pointed out elsewhere that if the reflexes which arise in the lung and which are expressed in the skeletal structures through the cervical nerves are produced through the afferent system which courses with the sympathetics in the same manner as those from the heart, stomach, liver and other organs, then Sherrington's law must be changed so as to read that each organ or tissue is connected segmentally in embryonic life with somatic structures in such a way that an afferent impulse arising in that organ or tissue will always discharge itself most readily through efferent neurons arising in those segments in the central nervous system with which it is bound embryologically.[1]

It is but natural to assume that the same laws that govern the production of reflexes in skeletal structures caused by stimuli arising in any given organ also govern reflexes which are produced in a given organ as a result of stimuli arising in skeletal structures. In other words, the segmental relationship which exists between an afferent visceral neuron and an efferent somatic neuron probably also exists between an afferent somatic and an efferent visceral neuron. From this we assume that there is a continuous flow of stimuli from the surface of the body inward to the viscera and from the viscera outward to the skeletal tissues.

Pulmonary Reflexes Expressed in Other Tissues and Organs.—One of the interesting and suggestive facts connected with the visceral neurology of the lung is the number of organs and tissues which are brought into relationship with the lung when it is the seat of inflammation. This I have studied particularly in connection with tuberculosis. The lungs being two separate and distinct organs of which one may be seriously diseased while the other remains comparatively free from pathology,

affords excellent opportunity for studying reflexes with a normal or near normal for comparison.

The vagus afferent fibers carry impulses from the lung to the medulla where they are transmitted to other branches of the vagus and to other cranial nerves to form reflexes in the same manner as the segmental reflexes are produced by spinal nerves in the skeletal structures as a

Fig. 2.—Reflex trophic changes in the tongue produced by stimulation of the hypoglossal nerve by stimuli which arise in a tuberculous lung and pass centralward over the vagus. The patient suffered from chronic tuberculosis of the left lung, the right lung being almost wholly free from disease. (From *Symptoms of Visceral Disease.*)

result of impulses which are carried from the viscera over the afferent system which is distributed with the sympathetic nerves.

Chronic unilateral tuberculosis offers an exceptional opportunity for studying such reflexes. I have described the atrophy of the facial muscles through the facialis; the atrophy of the tongue through the hypoglossal, as shown in Fig. 2; the atrophy of the mucous membrane

of the nose and pharynx through the facialis; and of the larynx through the laryngeal nerves. I have also suggested that changes in the pupil and in accommodation may be caused through the oculomotor nerve, and changes in teeth through the fifth nerve. Degeneration of the tongue and facial muscles occurs as a result of chronic inflammation of the lung. I have also been able to show an increased tension in those organs as a result of stimuli during the acute active stage of the disease, but this is sometimes difficult to demonstrate, especially in the tongue.[1, 8, 9]

Skeletal muscles, too, the sternocleidomastoideus and trapezius, may be reflexly affected through the vagus and the spinal accessory.[1, 8, 9, 11] Rasmussen[2] suggests that the many reflexes which I have described as affecting the skin, subcutaneous tissue and muscles supplied by the mid-

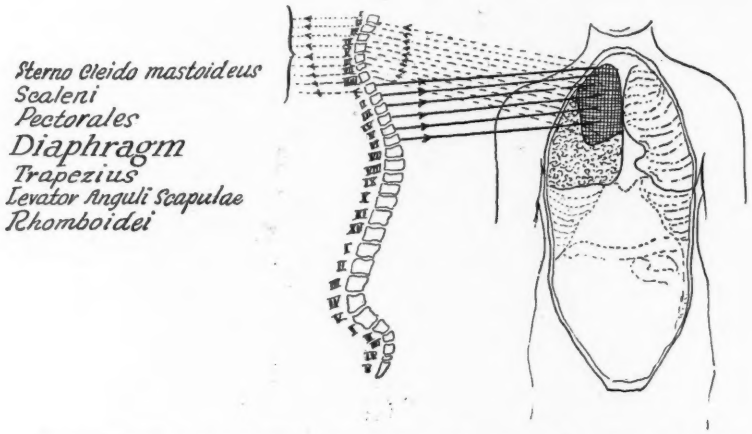


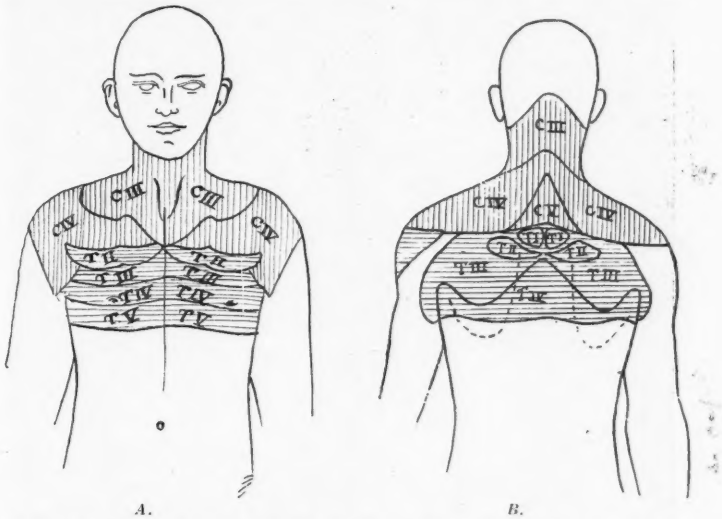
Fig. 3.—Schematic illustration of the pulmonary motor reflex in skeletal muscle. The impulse arising in the inflamed pulmonary tissues is carried centralward over the sympathetic system to the upper five or six thoracic segments of the cord, whence it is transmitted upward through intracentral paths to the midcervical segments and then transferred to cervical motor nerves, particularly C III, C IV and C V, to produce the motor reflex in the muscles innervated by these nerves, particularly the sternocleidomastoideus, the scaleni, subclavius, pectorales, trapezius, levator anguli scapulae, rhomboidei and the crus and central tendon of the diaphragm. (Rasmussen suggests the path might be through the vagus, the impulse being transferred downward from the medulla to the midcervical segments). (From *Symptoms of Visceral Disease.*)

cervical nerves may be caused chiefly by impulses which are conducted centralward over the vagus and transmitted downward in the cord to the midcervical segments. If this is true, I look upon it as a second path rather than the principal one.

The impulse may also be transferred from the afferent nerves of the vagus to the visceral fibers of the same nerve and so affect the motility of the larynx; increase the peristaltic and secretory function of the gastrointestinal canal, liver, and pancreas; and also cause inhibition of activity in the heart, resulting in its slowing.

The important organs of the thorax, abdomen, and pelvis are all capable of expressing pain in the skeletal tissues. In some this pain is excruciating, in others it is mild, but it is characteristic of the pain from each organ that its location is determined by the central segmental relationships of its afferent fibers. In other words, the impulse is carried to the cord and thus transmitted to skeletal sensory nerves of the same segment. In case the impulse is excessive, however, it may be transferred upward or downward in the cord or across to the other side, affecting neurons in segments other than the ones which it enters. This is seen in the spread of pain in angina and acute pancreatitis.

Impulses from these same organs likewise produce segmental motor reflexes in skeletal muscles. The impulse is carried centralward over the afferent segmental visceral nerves.



A. *B.*

Fig. 4. *A* and *B*.—Showing distribution of the pulmonary trophic reflexes as they are expressed in the skeletal soft tissues. The impulses arise in the inflammatory pulmonary tissue, are carried centralward, the same as in the pulmonary motor reflex, and are transferred to the cervical motor nerves C III, C IV and C V, to produce reflex degeneration in the muscles shown in Fig. 3, and also in the skin and subcutaneous tissue in the zones C III, C IV and C V, as shown in the cut. *A*. Anterior view. *B*. Posterior view. (From *Symptoms of Visceral Disease.*)

Since this system of afferent neurons which courses with the sympathetic nerves is present and recognized as the afferent system responsible for the visceral segmental reflexes and pain as expressed through the spinal nerves in case of all the other viscera; and since the reflexes from the lung are apparently identical in expression with these other reflexes except in segmental location, therefore, it seems justifiable to accept the same afferent system for the lung. This is all the more true since the vagary in its location can be accounted for on developmental grounds.

Fig. 5-*A*

Fig. 5. *A.* and *B.*—Showing the degenerative changes in the soft tissues of a patient suffering from extensive chronic tuberculosis of the right lung and pleura, and a slight lesion in the left lung.

Fig. 5 *A.*—Anterior view. On the right side it will be noted that the degeneration of the soft tissues extends from the angle of the law to the fourth or fifth rib. This indicates that both the underlying lung and pleura are involved.

The trophic reflex from the lung and visceral pleura is expressed between the angle of the jaw and second rib, which is very marked in this case.

The trophic reflex from the partietal pleura extends from the second rib down over the chest, at times even a little below the lower costal margin. Inasmuch as the degeneration is very evident between the second and fourth or fifth ribs, it shows that the parietal pleura is extensively involved.

Aside from the trophic changes it will be seen that the sternocleidomastoideus stands out somewhat prominently, which is indicative of increased tension due to the motor reflex, indicating an active inflammation in the corresponding lung at the time the picture was taken.

On the left side very slight degeneration is shown above the second rib. Apparently none below. This is indicative of a comparatively smaller healed or chronic lesion than that on the right, without any marked pleural involvement.

Therefore it seems reasonable to assume that those impulses which are responsible for the pulmonary reflexes expressed in the spinal nerves of the midcervical region as shown in the motor reflex in Fig. 3 and the trophic reflex in Fig. 4 A and B, and Fig. 5 A and B are picked up by the visceral afferent system which courses with the sympathetics, and that on entering the cord they are transmitted upward over intrasegmental neurons to the midcervical segments to the place where the lungs origi-

Fig. 5-*B*

Fig. 5 *B*.—Posterior view. It will be noted that the degeneration of the soft tissues extends down over the right shoulder and back. This is the result of both the trophic reflex from the lung, which expresses itself in the skin and subcutaneous tissue from the base of the skull to the spine of the scapula, and the parietal pleura, which expresses itself from the spine of the scapula down over the chest wall.

On the left side it will be noted that there is very little degeneration, it being practically confined to the areas above the spine of the scapula, indicating a smaller involvement than that on the right, but also chronic or healed.

The trophic changes are also marked in the interscapular space, indicative of an old chronic hilar lesion (hilar saucer of degeneration). These hilar tissues are extrapulmonary and show their reflexes in the interscapular region. (From *Symptoms of Visceral Disease.*)

nated in conjunction with the diaphragm, rather than to assume that they are of vagal origin.

REFLEXES EXPRESSED IN LUNG

Any reflex expressed in the lung must do one or more of several things: (1) it must contract the bronchial musculature; (2) it must relax the bronchial musculature; (3) it must increase bronchial secretion; or, (4) it must decrease bronchial secretion.

These effects are recognized as being present in certain well recognized pathologic pictures.

Asthma.—Asthma, for example, aside from the increased permeability of vessels and exudative phenomena, has certain bronchial muscle aspects; constriction and shortening, and an increase in bronchial secretion, as an important part of its clinical syndrome. It is a vagal syndrome.[12] It is now and then precipitated reflexly and there is no reason why the stimulus may not arise in the pulmonary tissues as well as in other organs.

Bronchitis.—Bronchitis, too, is a vagal effect, but one in which the chief influence is upon the mucous glands of the bronchi instead of the musculature. It is usually caused by local infection acting upon the bronchial glands, but a reflex mechanism is also present through which we may account for certain attacks of bronchitis caused by stimuli arising in the upper respiratory tract, the heart, or the organs of the gastrointestinal tract.

Pulmonary Collapse.—1. *The Picture.* Pulmonary collapse or acute pulmonary atelectasis, whether limited or massive, as we see it following abdominal operations, furnishes an interesting clinical picture. There has been much confusion connected with its explanation, but no matter where the stimulus which is responsible for the condition arises the primary bronchial effect is definitely a picture of excessive vagal action, thus: (1) a constriction of the bronchial lumen; (2) a shortening of the bronchial trunks; (3) an increase in bronchial secretion; and (4) an inhibition of inspiratory effort. This gives a clue on which to work in searching for the cause of this phenomenon.

The picture is modified by several other factors, some of which cause confusion, others of which may clarify. (1) Massive collapse is accompanied by an inhibition of activity of the diaphragm as a result of which it relaxes and assumes the position of paralysis similar to that which follows interruption of the phrenic nerve. (2) Very frequently, but not always, the collapse is on the same side as the abdominal pathology. However, it does seem to occur too frequently to be accounted for as a coincidence. (3) Collapse follows spinal anesthesia as well as general and probably occurs more frequently when it is used.

2. *Suggested Explanation.*—Every explanation of massive collapse so far offered has proved unsatisfying, therefore it seems wise to ap-

proach it simply as a picture to be analyzed, attempting to assess the part contributed by each important factor. We shall now attempt to discuss it from the standpoint of its being a vagal syndrome; for, of that, the evidence is definite, no matter how it is produced.

Vagal afferent fibers are distributed to all of the abdominal organs except the colon and sigmoid, and there is some question about the proximal colon. Vagal centers in the medulla, however, may be reflexly influenced by afferent stimuli carried over the sacral nerve, which together with the afferent vagal fibers, would bring the centers into reflex relationship with all important thoracic, abdominal and pelvic viscera. As previously stated, afferent vagal impulses from any viscus may express reflex activity through the pulmonary efferent vagal fibers, and by so doing, if the conditions are favorable, produce bronchial constriction and bronchial shortening, and an altered secretion of the glands of the bronchial mucous membrane, usually an increase in amount. Furthermore, afferent vagal impulses acting upon the respiratory center may produce inhibition of inspiration. These are the conditions noted in pulmonary collapse. It is also known that while stimulation of an afferent vagal fiber may act upon the respiratory center and produce either inspiration, or expiration (which carries with it an inhibitory effect on inspiration), it usually produces the latter. Thus the mechanism through which the stimuli necessary to produce reflex effects in the lung acts is present and could be called into play by operations which affect either the vagus, pelvic or any other mediating nerve.

In operations on the important viscera below the diaphragm ample opportunity for excessive irritation of both sympathetic and vagus afferent systems exists. In explanation of the vagal prepondèrance of action in the lung it must be remembered that the afferent sympathetic neurons from the abdominal organs which unite with efferent sympathetic neurons and antagonize vagal activity mediate in the lower six thoracic segments and not in the upper six from which the pulmonary sympathetics arise. Sympathetic reflexes from the abdominal viscera would express themselves by inhibiting muscular action in the gastrointestinal tract as in the ileus, which now and then occurs. So while the pulmonary vagal fibers are stimulated the antagonistic sympathetics are not, and so a condition favorable to over-vagal activity is brought about, which might be sufficient to produce the syndrome of collapse in certain individuals, particularly those of the vagotonic type.

Why collapse does not occur as a result of inflammation of these viscera without operation must be explained if the reflex theory is to hold, for we assume that afferent impulses course centralward over the vagus in all cases of inflammation of organs with vagal innervation. In case of operation it is not improbable that the opening of the abdomen, thus changing the negative pressure to a positive one and allowing the diaphragm to ascend following the contraction of the bronchi and the

consequent lessening of the lung volume is a most important factor in allowing mucus which has formed in response to increased vagal stimulation to accumulate in the bronchi.

The fact that the collapse does not always occur on the side of the organ operated upon, is in accordance with experimental work carried out by Dixon and Ransom,[13] in which cutting the vagus and stimulating the central end sometimes produced the effect in the opposite lung. There is also always the possibility of setting up irritation in other viscera than the one or ones operated on during the operation.

The *theory of the mucous plug* being the chief etiologic factor in the production of atelactasis and massive collapse seems to be held in greatest favor today, yet most students of the subject feel that it does not offer a complete and satisfactory explanation. It seems more rational to take it to be just one of many phenomena produced by some more remote causative mechanism. It surely will not explain those cases of collapse which in the World War resulted from a glancing ball which struck but failed to penetrate the chest; yet one British consultant, J. M. McDougall, told the writer that he saw four such instances. Cases have been reported, too, in which the bronchoscope failed to find a plug, but it was assumed that it had been dislodged by the patient.

By carefully analyzing and contrasting the two preponderantly vagal syndromes which are found in the lung, *asthma* and *pulmonary collapse*, data is brought forth which seems to me to clarify the subject very materially.

In asthma the bronchi are constricted and shortened. There is an increase in secretion of tenacious mucus, which is difficult to expectorate. The air cells are dilated, and if the lung collapses, it does so against great odds for the inspiratory effort is greater than the expiratory, which results in an emphysema and an increase in the size of the lung. The cough effort is increased. The diaphragm is not only held down by the negative intraabdominal pressure but is actually pushed down by the dilated air cells and the increased positive intrathoracic pressure. Because of these facts opportunity for an extensive plugging of the bronchi rarely exists. Furthermore, the abdominal muscles and all other muscles of respiration, both normal and accessory, are brought into play as a result of the increased CO_2 caused by the prolonged deficient areation of the blood. The abdominal muscles, too, are uninjured, and consequently are able to meet the increased work thrown upon them both by respiratory demands and the demands of the cough reflex for keeping the bronchi free.

In pulmonary collapse the formation of tenacious mucus is also increased, but its retention is favored: (1) by the constricted and shortened bronchi; (2) by the collapse of the lung permitted by the reduction or elimination of the negative intraabdominal tension, and

the consequent ascent of the diaphragm, allowing the lung to lessen in size following the contracted bronchi; (3) by the failure of the abdominal muscles and other normal and accessory muscles of respiration to be called into play sufficiently to clear the bronchi of secretion, because of insufficient CO_2 stimulation; and (4) by an insufficient cough reflex. The cough reflex is probably lessened or eliminated by the collapse of the tissues if we may gauge by the relief of cough which follows pneumothorax in tuberculosis.

That the mucous plug is usually present and *that relief usually follows its removal, and forced respiration,* and sometimes either alone cannot be denied, but this furnishes incomplete evidence of the etiologic rôle played by the mucous plug. We have increased mucus in the vagal syndrome of asthma, yet plugging of the bronchus and collapse of tissues rarely result; therefore it seems reasonable to look for the cause of the difference in effects in these two conditions in other accompanying manifestations, as mentioned above. The fact that collapse may recur for several days but not after the patient has recovered from the immediate effects of the local injury is also suggestive of its nature.

In explanation of the fact that massive collapse follows spinal anesthesia for operations on abdominal viscera quite commonly, attention should be called to the ease with which the anesthetic may ascend in the cord and paralyze the sympathetic connector neurons in the upper six thoracic segments, or at least in the lower of these segments, thus removing their antagonism from the reflexly stimulated bronchial branches of the vagus and permitting a vagal preponderance to occur.

The fact that vagal preponderance, unaided, is not sufficient to produce collapse of the lung is shown by Cannon's cats which maintained normal respiratory function after all opposing sympathetic antagonism was removed by complete removal of the sympathetic system. But here no excessive vagal stimulation was present.

In the conditions which we are discussing it might be that collapse is more apt to occur in certain individuals who may have hyperexcitable vagus nerves, for we do recognize the vagatonics as belonging to a class which reacts differently from those with more stable nerve balance; or, it may be the same as we find in all diseases, that various persons show different symptoms to what to us seem to be similar stimuli. These persons may react in this manner simply because of certain peculiarities in their own physiologic make-up. They are thrown into the class with preponderant vagal action, as a result of the operation; and the collapse is a secondary affair which takes place because of the opening of the abdomen and the conversion of the intraabdominal negative pressure into a positive pressure, and the plugging of the bronchus is due to lack of an excess of CO_2 and the cough reflex necessary to keep the bronchial lumen free.

For those cases which were caused by a glancing missile in the war the most reasonable explanation seems to be a reflex produced by stimulation of the afferent nerves of the superficial structures covering the chest wall, plus excessive fright at the time of its occurrence. All the accessory muscles of respiration, including the pectorales and broad muscles of the back, are innervated by an afferent system which carries the impulses backward to the same midcervical segments of the cord with which the lung is embryologically connected, as previously mentioned. This then, through intracentral paths, furnishes a basis for mediation in the upper thoracic segments from which the pulmonary sympathetics arise. The regular effect of such a stimulation would be to increase sympathetic effects and decrease vagal effects; but we have a hint as to how an opposite effect might be produced by the involuntary bowel movement which is experienced by soldiers in battle. While the emotion of fright acts upon the sympathetic nervous system, as has been demonstrated so convincingly by Cannon, and would have a tendency to contract the sphincters, severe fright might cause a temporary paresis and inhibition of the sympathetic nerves and so cause an opposite effect by leaving the parasympathetics unopposed. This too would be aided by the extra reflex stimulation.

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Department of Reviews and Abstracts

CONDUCTED BY DUFF S. ALLEN, M.D., ASSOCIATE EDITOR

Selected Abstracts*

Physiology

Luisada, Aldo. La Contractilité Active du Poumon etudieé au Moyen de L'electro-bronchographie. (Active Contractility of the Lungs Studied Through Electro-bronchography.) Arch. méd.-chir. de l'app. respir. 5: 320, 1930.

The clinical conceptions of the elasticity of the lungs have not been satisfactory or sufficient to clarify some of the pathologic problems that come up in the course of pneumothorax, hydrothorax, emphysema and asthma. The following study came about quite by accident in observations in oncographic studies on cats. A lung, twenty minutes postmortem, while in contact with a Marey's apparatus, was observed to display rhythmic, systolic and diastolic motion. In reproductions of this phenomena it was noted that it could be exhibited only rarely spontaneously, but more frequently when the lung was exposed for observation just prior to an expansion. The subsequent contraction following bronchial distention was combined with the elastic recoil of the lung. The smooth muscles showed following expansion a quickly set, slowly relaxing, strong tonic contraction which then changed slowly into the preceding rhythmic clonic contractions.

To study exactly the actions of this bronchopulmonary musculature determinations of its action current were undertaken.

The following were noted with isolated cats' and rabbits' lungs: At rest there is a slow or lively, mono- or biphasic current which can go on for hours at a time. The phenomena varied clearly and consistently with the rhythmical expansion of the lungs which acted as the active stimulus for the smooth muscles. The amplitude of the electrical waves was proportional to the degree of expansion as long as this was held within physiologic limits. The action currents were appreciably greater when the pauses between the pulmonary expansions were longer. The most advantageous lead-off electrode for the current is between the trachea and one of the large bronchi.

Further studies on the living animals were performed wherein one electrode was introduced into a small bronchus of the lower lobe while the other lead off the apex to be plunged into normal saline. To render the action currents of the heart ineffective the filter of von Pasoli was employed. Simultaneous pneumotachograms and records of abdominal respirations were registered. The term electrobronchography, analogous to electrocardiography, is given to the registered total of the action currents of the bronchopulmonary smooth muscles obtained by this method. Its amplitude stands in direct proportion to the depth of respiration and in inverse proportion to its frequency. With consistency of the depth and of the frequency of respiration there is shown an increased upstroke in the electrobronchographic curves,

*The abstracts in this issue were compiled by Dr. M. M. Berck, Research Fellow in Thoracic Surgery, Washington University School of Medicine, St. Louis.

showing an increased exertion on the part of the smooth muscle. Electrobronchography was also determined upon two hundred human subjects. The following technic was used: Under anesthesia of the upper respiratory tract, one electrode was inserted into the bronchial tree and another was placed around the neck. The electrical records received exhibited curves of the action currents of the bronchopulmonary musculature. There were no untoward disturbances of any sort. The normal EBG of man shows great individual variations. Frequently in the middle of expiration a biphasic wave appears which signifies a quick energetic contraction of the bronchial muscles, but this was not regularly encountered. Occasionally an increment in the waves through synchronization with the EKG current occurs. The EBG thus allows one to ascertain changes in tonus and contractility of the bronchopulmonary musculature.

Further elucidation is secured through combination of EBG and pharmacologic studies. Ether (per rectum) by a synergistic action upon thoracic and abdominal striated muscle and the expiratory phase of bronchial contraction exerts a beneficial expiratory effect. A dysynergy between smooth and striated muscle is found with large dosage of histamin, in anaphylactic shock and after vagus section. Large doses of atropin and papavarin bring the EBG to the vanishing point with the abolition of respiratory contractility. With histamin there is obtained the strongest EBG wave indicative of the dysynergy appeared by this drug between the striated and the bronchopulmonary smooth musculature. Acetylcholin, by comparison, demonstrates: (1) Initially a diminution in the amplitude of the EBG as the first expression of hypertonus of the smooth muscle, (2) the second phase is an increase in the strength of the waves with a prematurity of the electric waves (this phenomenon is shown by no other substance and serves to make a distinction between histamin and acetylcholin in organic fluids), (3) the third phase is that of hypercontractility. Adrenalin in small doses diminishes the amplitude. In large doses it exhibits phenomena similar to acetylcholin and histamin so that in all likelihood these drugs act through an adrenalin action.

The influence of the vagus was studied with bilateral vagus section and with direct, chemical, central and reflex stimulation. The results point to a controlling influence of the vagus upon the muscular activity of the lung and a probable relation to tonus. Sympathetic stimulation results in a slight flattening of the electrical waves. Experiments on the phrenic show that fibers in these nerves can stimulate the smooth bronchopulmonary musculature. Experiments in anaphylactic shock with dogs, cats, rabbits and porpoises, show an increase in bronchopulmonary contractility, and at the same time a loss of synergy between the smooth and striated muscles.

The findings with pneumothorax seem important: Unilateral total pneumothoraces were induced in dogs, cats and porpoises through openings to the outside air. The opposite side then exhibited a definite increase in contractility. The collapsed lung, however, breathes through movements of its smooth muscle in synchronous rhythm. Furthermore its curve coincides with that of the other side almost completely. Bilateral vagus section or extirpation of the stellate ganglia had no influence on the results with pneumothorax. In contrast with this bilateral phrenic section prevented the marked increase in contractility, of the bronchopulmonary musculature usually present on the opposite side in the pneumothorax experiments. Therefore the conclusion is drawn that the stimuli for increase in lung contractility with pneumothorax and reflx phenomena go from the pleural endings by a centripetal route through afferents in the phrenic nerve while the efferents probably are represented by sympathetic fibers not running through the stellate ganglia, but, more probably, coursing in the route of the phrenic.

Reinberg, H., and Kopziovskaja, Zatkin: Massiver Lungenkollaps; Experimentelle Untersuchungen. (Massive Pulmonary Collapse; Experimental Studies.) Deutsche Ztschr. f. Chir. **230**: 182, 1931.

The authors report 20 dog experiments on the production of pulmonary collapse. The method used was the plugging of bronchi with laminaria tent after performance of a tracheotomy. All the animals died within two hours to five days. Autopsies show that there appears in many cases true pneumonia on the basis of the collapse with frequent transition to suppuration. The results as noted by others of mediastinal displacement dependent upon the degree of mobility of the other intrathoracic structures were likewise noted. In none of the experiments could the re-expansion of the lungs be secured after withdrawal of the bronchial plug. This is contrary to the results of other authors. The authors stress the importance of the findings that the collapsed shrunken lobes were always markedly engorged and congested. They ascribe this to the aspirating action upon the blood vessels and lymphatics exerted by the negative pressure of the collapsed lobes. In this respect collapse induced by bronchial blockage is to be differentiated from that due to pulmonary compression (as from large pleural effusion) in which the blood is expressed from the lung, rendering it ischemic in contrast with the collapsed lung due to bronchial blockage in which the authors found marked hyperemia. The authors state that the presence of this marked congestion may serve to clarify the understanding of the pathological changes that follow bronchial obstruction and pulmonary collapse. Almost all postoperative pulmonary complications are thought due to pulmonary collapse in various anatomical localizations, being lobar or scattered and patchy.

Adams, W. E., and Livingston, H. N.: Obstructive Pulmonary Atelectasis. Arch. Surg. **23**: 500, September, 1931.

Recent publications have described two etiologic factors as being essential to the production of pulmonary atelectasis; viz., bronchial obstruction and labored respiration, or respiration against resistance. The latter of these observations was contrary to the experience of those who had produced the condition experimentally with regularity with a quiet shallow respiratory cycle.

The experiments made use of an obstructive mechanism that was entirely physiologic in the production of the results. They aimed at determination of the effects of different localizations of the obstruction and the effects of the rôle played by different types of respiration. Dogs were used. Cauterizations of the bronchi with 35 to 50 per cent solution of silver nitrate was performed by bronchoscopy producing gradual complete stenosis of the bronchi with resultant atelectasis. The number of applications of silver nitrate varied from one to three, and the time consumed in producing complete atelectasis varied from one to six weeks depending upon the diameter of the bronchus and the strength of the silver nitrate solution.

It has been generally accepted that the production of massive atelectasis, clinically, is favored by the presence of a shallow respiratory cycle. It is contended that this form of respiration leads to the collection and accumulation of secretions within the air passages.

If an obstruction is so located that all of the lobe is affected, complete atelectasis follows. However if a small amount of a lobe is left unobstructed, no atelectasis is produced. It appears probable that there are communications between the lobules of a single lobe. This explains why collapse is not obtained when a part of a lobe is left unobstructed.

The type of respiration did not seem to play an important rôle in producing atelectasis. Massive pulmonary atelectasis could always be produced in the presence of normal respiratory excursions. Indeed, clinically, shallow respirations do not seem to be of such capital importance as has been ascribed to them since many patients are seen who have shallow respirations postoperatively yet never develop pulmonary atelectasis, and not uncommonly patients with normal respirations develop massive atelectasis.

Clinically two types of atelectasis can be described according to the rates of production.

1. Certain cases, occurring within a few minutes after operation, do so by means of a check-valve bronchial obstruction aided by a straining type of respiration.

2. Another group is that in which the atelectasis developed slowly over a period of hours accompanied by no particular type of respiration, probably caused by a progressive type of occluding bronchial obstruction such as was produced by the experimental cauterization of the bronchi.

In conclusion:

1. Complete lobar obstruction is essential to the production of atelectasis.

2. Lobular obstruction is not followed by atelectasis of the obstructed part.

3. No particular form of respiration seems essential to the production of atelectasis except that a staining type of respiration in the presence of a check-valve obstruction seems to hasten its production.

4. Pulmonary suppuration was not associated with obstructive pulmonary atelectasis produced experimentally in dogs in spite of the fact that the parenchyma of normal dogs and of atelectatic lobes is always infected with bacteria (the same types in both instances).

Blavier, L.: Mesure du collapsus Pulmonaire Experimental (Experimental Measure of Pulmonary Collapse). Arch. internat. méd. expér. **4**: 270, 1930.

To facilitate the understanding of certain pathologic phenomena occurring in cases of pulmonary collapse, experiments were undertaken to secure precise measurements of the degree of collapse: (a) The degree of unilateral pulmonary collapse, (b) the difference in collapse between the upper and lower portions of the collapsed lobe, (c) the relative degree of the collapse of the upper and lower portions of the side opposite to that collapsed.

The variations in pressure in the pleural space reflect those existing in the neighboring lung—therefore the technic involved the measurements of these pressures. To measure the degree of collapse a collapsible balloon was introduced through a closed system of telescoping tubes into the pleural cavity. The procedure employed obviated the formation of a pneumothorax. Manometric measurements could thus be taken of the pressures exerted upon the balloon.

To obtain collapse phrenicectomy was performed. This was followed by a great diminution of all the variations in pressure above and below within both pleural cavities. This diminution in variations of pressure was most marked at the apex of the operated side, a little less marked at the base of the operated side, less at the non-operated apex and least at the non-operated base. The collapse due to phrenicectomy appears, therefore, to affect the apex quite as much as the base. The effect on the contralateral side may be due to deviation of the mediastinum.

Chest Wall

Kaufman, R.: Quelques considerations sur l'abces froid thoracique. (Cold Abscess of the Thoracic Wall.) J. d. chir. 37: 829, June, 1931.

Tuberculous abscesses of the thoracic wall formerly have been thought to originate from (a) osteitis of the rib, (b) the parietal pleura, (c) adenitis of the internal mammary chain. The dissection of operative specimens seems to demonstrate the origin of the tuberculous infection to be always in the lymph nodes lying in the intercostal spaces. When osteitis is encountered, it is constantly in that portion of the sternum or ribs directly in contact with the universally present tuberculous adenitis and contiguously secondary to it. Chondritis is rare. Resection should include the retrocostal tuberculous glandular mass in addition to the excision of the subcostal abscess and the diseased rib. Success is not usually obtained by rib resection alone for supposed tuberculous osteitis. This is due to the failure to remove the caseous nodes which had caused tuberculous erosion of the rib at the site of contact and had always been antecedent to it. The pleura at the time of excision of a cold abscess usually appears grossly healthy. However, it is believed that a cold abscess is always a sequel of an antecedent tuberculous pleurisy usually healed at the time of observation. The internal mammary and laterovertebral lymphatic chains draining the collateral pleura become infected at the time of the tuberculous pleurisy and the adenitis is therefore pleurogenic. This tuberculous adenitis can go on to abscess formation and suppurative peripleuritis, and if contiguous to bone, it brings about tuberculous osteitis. An exception to this is the cold abscess formed along a puncture tract of a tuberculous pleural collection.

The operative technic advised is wide resection en masse of all the contiguous area down to the pleura. Small abscesses are excised en bloc and the defects sutured over, large abscesses with fistulas are packed open after block excision. Modifications of the technic are described for laterosternal, dorsal, abdomino-thoracic and fistulous abscesses.

Rehn, H.: Zur Chirurgie des Brustbeins und Physiologie des vordern Mittelfells. (Surgery of the Sternum and Physiology of the Anterior Mediastinum.) 55 Tag. d. Deutsch. Ges. f. Chirug. Sitzg. 8.-11. IV. 1931.

The sternum by its structure and configuration determines the type of respiration, besides which, it serves to protect the heart and great vessels. In those animals in which the sternum is absent the pericardium is much thickened, stiffened and thus takes over the task of the protection of the heart and great vessels. In operative conditions involving the sternum a clear distinction must be made between the upper and lower portions of it. The lower portion can be removed without any disturbance whatsoever arising from the extirpation. However the phenomena following the removal of the upper sternum demand serious consideration. The upper portion of the sternum lies in closely knit, tissue-bound, union with the heart, the great vessels and the tissues of the mediastinum. The sternopericardial ligament running from the inner surface of the upper portion of the sternum to the superior vena cava and the right heart has a very important function; i.e. it serves to keep the great venous spaces expanded and patent. The normal aspiration of the thorax and right heart extends its influence to the furthermost veins. The prevention of reflux requires the action of the thoracic bony structures and ligaments to keep the venous spaces patent. With extirpation of the upper portion of the sternum one also loses the upper ribs through loss of their insertions. A sequel to removal of the sternum therefore may be the reversal of venous flow with a tremendous lowering of the volume filling inflow into the right heart eventuating in cardiac standstill

and exitus. Milder forms may be manifest as circulatory depression and circulatory failure with cyanosis. These highly dangerous states may come on very shortly after extirpation of the upper sternum. To guard against this the author has devised a technic of the substitution of a free tibial graft for the extirpated upper sternum. Postoperative x-rays show that with this method of support the important function of the ligaments attached to the sternum through renewal of their bony insertions is preserved and that the mediastinal spaces are kept patent. This greatly facilitates such operative procedures as removal of substernal thyroids and other neoplasms in the anterior mediastinum. Intravenous injections of acacia—Ringer's solution (6%) aid in keeping the venous spaces of the mediastinum filled during operation.

Pässler: A great similarity exists between the sternum and the vertebral column. The intervertebral discs correspond to the cartilaginous union between the manubrium and the sternum, a similarity seen also in pathological studies. The calcareous infiltration and ossification of the two cartilaginous structures can be very similar. Usually, however, ossification does not take place between the manubrium and the body of the sternum. Even in the very old only 7 per cent of the cartilaginous unions were ossified. Osteitis deformans (Paget's) was present in 21 per cent of the cases of fracture and dislocation of the sternum seen. Usually only the body or the manubrium was affected but occasionally both were involved.

Mediastinum: Pericardium and Heart

Gibbon, J. H., and Churchill, E. D.: The Mechanical Influence of a Pericardium Upon Cardiac Function. J. Clin. Investigation **10:** 405, June, 1931.

The action of the heart may be profoundly disturbed by alterations in the pressure maintained over its outer surface. Familiar examples are fluid cardiac tamponade and "concretio pericardii" of Pick's disease which is a chronic tamponade acting through a contracted and scarred pericardium that tightly grasps the heart as with a vise, impeding diastolic filling.

Previous literature by other authors has variously noted:

1. The pericardium can inhibit a great acute dilatation of the heart.

2. Opening of the pericardium had a favorable influence in acute experimental mitral and tricuspid valvular defects but not in experimental valvular defects of several weeks' duration.

3. Experimental removal of the pericardium is compatible with active life.

4. Congenital pericardial deficiency cases are not appreciably altered from the normal as far as cardiac efficiency is concerned.

In this investigation the following were studied:

1. The effect of opening the pericardium upon function of the normal heart as indicated by systolic and venous blood pressure.

2. The effect of opening the pericardium when the work of the heart has been greatly increased by the partial obstruction to the outflow of blood from one ventricle.

3. The effect of removing a constricting pericardium in the presence of a partial occlusion of the pulmonary artery or aorta.

4. The tamponade effects produced by increasing the work of the heart within a snugly adjusted pericardium. The technic employed entailed exposure of all the intrathoracic structures to atmospheric pressure with a consequent diminution of cardiac output and presumably a slight decrease in diastolic heart size.

The results summarized are:

1. Opening the pericardium is without effect upon either the arterial and the venous pressure in the normal heart of the cat.

2. Gradual compression of the pulmonary artery results in a slow fall in arterial pressure and a corresponding rise in venous pressure.

3. Opening the pericardium when cardiac decompensation has been produced by compression of the pulmonary artery or aorta is without significant effect on the elevated venous pressure or the low arterial pressure.

4. Tight closure of the pericardium with sutures produces cardiac tamponade with a fall in arterial pressure and a rise in venous pressure. On opening the pericardium again the pressures return to their former levels.

5. When the pericardium has been closed with the production of cardiac tamponade and partial obstruction of the pulmonary artery has been superimposed, opening the pericardium causes a rise in the carotid pressure and a fall in the superior caval pressure.

6. By exercising considerable care it is possible to close the pericardium with sutures without affecting the arterial or venous pressure. Subsequent opening of the pericardium is without effect upon the pressure levels.

7. When the pericardium has been so closed without affecting either the arterial or venous pressure and cardiac decompensation has been produced by compression of the pulmonary artery, opening of the pericardium causes a rise in arterial pressure and a fall in venous pressure.

8. In none of the experiments was removal of the pericardium detrimental to the circulation, even when signs of cardiac decompensation had been experimentally produced.

9. Increased work is accomplished by increase in the diastolic size of the heart. Removal of the pericardium which ordinarily limits this normal cardiac dilatation materially increases the degree of cardiac dilatation.

Decompression of the heart may, therefore, in favorable instances, enable it to compensate more adequately for valvular defects.

Fischer, H.: Die Bedeutung der Kardiolyse fur die Therapie. (The Importance of Cardiolysis in Therapy.) Fortsch. Ther. 1930, p. 557.

The author summarizes the possibilities and indications in cardiac surgery and discusses a proposed alteration in the operatve technic of the cardiolysis of Schmeiden with the report of a case. When adhesions of the external pericardium are present binding it to the anterior chest wall and tethering it to the diaphragm, Brauer's cardiolysis is recommended which effects the removal of the anterior bony chest wall. Fischer suggests the name of precardiac thoracolysis for this procedure.

When adhesions are present between the heart and the pericardium, the mere separation of them has occasionally met with success, but decortication procedures prove to be the most reliable. The author reports a case performed according to the Schmeiden technic which had disastrous results. Although Schmeiden has reported several cases with gratifying outcome, the author feels that this case will serve to point out a danger possible in the procedure. In a case of adhesive pericarditis, the freeing of the right ventricle was effected after the clearing off of the adhesions of the right auricle and left ventricle. At the end of this procedure the following was noted: The liberated right heart enlarged progressively, swelling and ballooning out, the cardic action grew weaker and finally under observation came to a standstill in spite of stimulation. Histologic examination later showed marked scarring and degeneration of the musculature of the right heart. It is

supposed that with the freeing of its fetters the right cardiac muscle had lost its tone. On the basis of this experience, the author proposes alteration of Schmeiden's procedure in this respect—that only the strongly musceled left ventricle be decorticated and freed. This restricted operation is a much less perilous procedure and the results of the author with it have been favorable.

Patients should come for operation much sooner than they have hitherto to avoid the progression of the cardiac muscle degeneration.

Grossman, M.: Zur Differentialdiagnose der Herzbeutelverwachsung. (The Differential Diagnosis of Adhesive Pericarditis.) Schweitz. med. Wchnschr. **61**: 372, 1931.

The usual difficulty in the diagnosis of adhesive pericarditis lies in the fact that the clinical picture produced by it is also evoked by other diseases; namely, a progressively weaker cardiac action. Even the so-called diagnostic sign—apical systolic retraction of the chest wall, is not a constant finding in a number of true cases which never show this sign. The pathologic findings at pericardiolysis have revealed a great disproportion between clinical diagnosis and operative findings. Volhard has classified the subject with the description of two main types. The first rises through gross adhesions between the pericardium and surrounding structures and so by its adhesions to the anterior chest wall causes the well-known apical systolic retraction. Its encompassing hindrance to systolic action is the explanation of the progressive diminution of the cardiac power seen in these cases. Pericardiolysis may produce very beneficial effects in these cases. In the second group through the thickening and advanced contracture of the epicardium and pericardium there is produced a great hindrance to diastolic filling—a stasis promoting influence producing the severe symptoms of a "stasis syndrome." The operative treatment in these cases is not so successful as in the first group. The author demonstrates a series of cases in which this stasis syndrome is produced by intrathoracic factors other than pericardial adhesions. These lesions were confined processes in which there were infiltration, thickening, cicatrization and later contraction. In the main they are inflammatory processes in the wall of the heart, in the mediastinal structures and in the mediastinal pleura. Processes of a nature akin to pericarditis can in these structures produce the stasis syndrome through pressure upon the great vessels entering the right auricle. Operative resection and freeing of these adhesions will lighten the circulatory burden. Therapy in these cases should include other measures such as diuretics.

Leriche, R., and Bauer, R.: Tumeurs Primitives du coeur (Primary Tumors of the Heart.) Arch. d. mal. du coeur, **23**: 645, October, 1930.

The basis of this report is a case of cardiac sarcoma, which completely filled the right auricle and yet never gave any circulatory symptoms. The patient (female of fifty-three) was operated upon for a mediastinal tumor, approximately the size of two fists, which was in reality but an extension through the pericardium of the cardiac neoplasm. The tumor could not be removed and the patient ceased soon after operation.

There are only 150 observations of cardiac neoplasm in the literature. The most frequently seen are myxomas, fibromas, lipomas, sarcomas and rhabdomyomas. Three types are distinguishable, tumors of the parietes, intracavital and valvular.

The tumors of the valves are usually small pedunculated neoplasms which histologically are myxomas or fibromas. Often pseudomyxomas derived from organized thrombi are found.

The intracavital tumors are more frequent. They lie most commonly in the left auricle and appear either as pedunculated tumors floating in the cardiac cavity or, becoming adherent to the endocardium, assume a sessile form and end by filling up the auricular or ventricular cavity. Primarily they are comparable to valvular tumors, secondarily they assume the characteristics of parietal tumors.

Tumors of the wall are extremely variable in their sites, size and histologic make-up. They appear most frequently in the auricular walls and most commonly in the auricle. Usually composed of small nodules they can, however, attain large dimensions. When of large size they can deform the auriculoventricular orifices and compress the great vessels. Tumors developing in the interventricular septum can compromise the bundle of His. These tumors are represented by two main histologic types—sarcomas and rhabdomyomas. Much more exceptional are fibromas, lipomas and endotheliomas.

In spite of the anatomic site of these tumors the clinical diagnosis has never been made. The symptoms are indeed not at all characteristic. Tumors of the valves present valvular signs, dependent upon the site and size of the tumor either of stenosis or of combined lesions.

Pedunculated intracavital tumors can be absolutely silent even though voluminous, if they are situated at some distance from the valvular orifices. If they arise near the valves they compromise the orifices. They can, in certain cases, suddenly become engaged in the orifice, obturating it with sudden death.

Partial tumors give the least characteristic signs because its cardiac manifestations are usually negligible. The following signs have been occasionally observed; tachycardia, arrhythmia, slight cyanosis of the face, due to compression of the superior vena cava, and often signs of enlargement of the heart—with increase in width and displacement of the apex. However, in certain cases cardiac signs dominate the scene—asystole with stethoscopic signs of complete stenosis of an auriculoventricular orifice when the tumor suddenly obturates the orifice and Adam's-Stokes syndrome when the tumor is in the interventricular septum and compromises the bundle of His. X-ray does not help in the diagnosis. If the tumor is very large the appearance is that of a large mediastinal tumor with displacement of the heart.

Mediastinum: Thyroid, Neoplasm

Lahey, F. H.: Surgical Management of Intrathoracic Goiter. Surg. Gynec. and Obst. **53**: 346, September, 1931.

Intrathoracic goiters may be divided into completely intrathoracic and incompletely intrathoracic. In the completely intrathoracic type, the greatest diameter of the goiter is below the top of the sternum and in the incompletely thoracic type the point of greatest diameter is above the suprasternal notch. Most intrathoracic goiters are adenomata and most completely intrathoracic goiters are adenomata or cysts. The explanation of the intrathoracic position of adenomata is the enlargement of an adenoma of a lower pole downward, its course unopposed, forced by the swallowing movements to mold a pathway into the superior mediastinum. After the adenoma becomes so great in diameter that it can no longer pass back through the thoracic aperture, it becomes completely intrathoracic. Once intrathoracic its growth may extend the tumor to lower levels. Projections of adenomatous thyroid tissue starting from one or both inferior thyroid poles may extend moderately or deeply into the thoracic cavity.

The symptoms produced by the intrathoracic goiter are for the most part the results of pressure upon the trachea. This may be so very gradual as not to be

brought to the patient's attention although noticeable to others. A dyspnea in different positions may result from angulation of the trachea from the alterations in position of the intrathoracic adenoma. Occasional bouts of threatened suffocation may occur probably due to accumulation of mucus in the trachea below the point where the trachea is narrowed by the intrathoracic goiter. A violent struggle to breathe with the expulsion of the plug of mucus terminating the attack of suffocation is the history occasionally given.

Deviation and lateral torsion of the trachea is often seen by x-ray. In most instances the goiters are located to one side of the trachea with the trachea curving around the portion of the goiter against which the trachea rests. The trachea, however, may be flattened from before backward by a central goiter. Dilatation of the superficial thoracic veins over the upper chest wall is common.

In the removal of intrathoracic goiter—ligation and severance of the superior vascular thyroid pole permits mobilization of the upper portion of the tumor so that its pole may be turned forward thus exposing the posterior wall of the thyroid and the point where the great vessels are in contact with it. The goiters should be removed intact since fragments left behind become necrotic and predispose to infection and mediastinitis. Deliverance of the goiter should be performed by digital pressure from below upward with tension at the upper margin of the tumor to elongate it and render it of diameter small enough to pass through the thoracic aperture. This has made possible the delivery of some intrathoracic adenomata that would otherwise necessitate splitting of the sternum. If the degree of tracheal obstruction is considerable, before removal of the intrathoracic mass, a tracheal catheter should be passed, so that with positive pressure danger of suffocation during delivery of the tumor is overcome. Ligation of the inferior thyroid artery is rarely necessary since the artery is elongated by the gradual descent of the tumor. The adenoma can be removed preserving a posterior shell of thyroid tissue which insures preservation of the lower parathyroids and recurrent laryngeal nerves. It is quite important to control any oozing veins, since the accumulation of a hematoma within the mediastinum predisposes to infection and mediastinitis. The cavity should be packed lightly but completely filled. This produces protective mediastinal granulations which wall off the space if secondary accumulations of infected serum should occur in the mediastinal cavity. The cavity should be kept open with gauze or cigarette drains until it has completely collapsed and filled up from the bottom so that serum cannot accumulate.

Patients with low-lying adenomata of the thyroid should be kept under observation. Should the adenomata show downward progress they should be removed.

Middeldorpf, K.: Ueber Geschwülste des Mittelfells. (Mediastinal Tumors.) Deutsche Ztschr. f. Chir. **228:** 43, October, 1930.

The article is based upon 15 cases of mediastinal tumors operated upon by Sauerbruch. The pathologic varieties were lymphosarcoma, carcinoma of substernal thyroid, aneurysm of the aortic arch, chondroma of the vertebral disc, and retropleural lipoma.

The mode of approach depends upon the site of the tumor. In cases of intrathoracic thyroid carcinomata, Sauerbruch branches off on the collar cervical incision to continue on a median sternal incision for a sternotomy. In cases of anterior mediastinal tumors the approach is through a median sternotomy or a parasternotomy with resection of the parasternal costal cartilages. In cases of posterior mediastinal tumors the approach is through a posterior mediastinotomy with paravertebral rib resection. In cases of severe compression caused by mediastinal tumors with dyspnea, dysphasia, compression of the vena cava, Sauerbruch

has discarded tracheotomy for the relief of dyspnea. He prefers to decompress the thorax by a median sternotomy or resection of the sternum. Ten such decompressive sternotomies have been performed. Each time the patient has been considerably relieved and able to live without great discomfort for several months.

Mediastinum: Esophagus

Woodman, E. M.: Treatment of Carcinoma of Esophagus. Brit. Med. No. 3684, p. 290, August 15, 1931.

The principal difficulty in the treatment of the carcinoma of the esophagus lies in the anatomic formation of the esophagus. Suspended at the entrance, where it is attached to the back of the cricoid cartilage, and fixed at the lower end, where it traverses the diaphragm, it remains completely mobile in the center. In the cervical region the gullet lies in an intimate contact with the trachea, into which perforation readily takes place. In the central zone the left bronchus is easily invaded. In the lower portion, the pleura is wrapped around the gullet and damage or infiltration readily takes place; while in the diaphragmatic region vascular and lymphatic anastomosis is so free that the condition is hopeless from the outset. The absence of a peritoneal coat is of vital importance. Such a coat, if present, would delay extension and give rise to valuable adhesions. Injection experiments of the draining lymphatics of the esophagus have shown that a most widespread dispersion can take place involving the glands at the base of the neck and the glands of the mediastinum and intercostal spaces. A large extension can take place on the parietal and diaphragmatic pleura. The initial lymphatic spread may be slow and go on with difficulty but once the primary barriers are broken through, the invasion is widespread and apparently rapid.

Diathermy. This procedure is capable of complete destruction of a localized growth but has to be used with care or fatal operative results take place. To operate with safety by diathermy it is necessary to establish a connective tissue reaction outside the growth. This has not met with success. Diathermy is a painless operation and results in dilatation of the lumen of the gullet and increased ease of feeding. But it is not without danger and several patients have died within a day or two of the operation from leakage into the pleural cavity. As a curative means it has proved a complete failure but for palliation it is of distinct value.

Radium Treatment. (A) External treatment. Few cases of carcinoma of the esophagus within the neck present themselves for treatment. In these few cases a large proportion consists of that peculiar difficult growth which takes place within the sphincter at the entrance to the esophagus behind the cricoid. In general if the growth can be reached in the neck experience is all together in favor of excising it in any way possible. The external application of radium has not proved of much value.

The great drawback of the external approach to the esophagus with a view to implanting radium is that such treatment would of necessity have to be repeated.

(B) Interstitial application. By this means radium emanation is carried into the substance of the growth. The position of the lumen is ascertained and if necessary it is dilated. The radon seeds are inserted by a trocar and cannula. The seeds are buried deeply into the upper part of the growth, their number and position are checked by x-ray examination on the following day, at the end of the week, and at the end of the month. The disadvantage is that only the upper part of the growth

can be dealt with at the first operation, leaving the lower part to be dealt with at a subsequent insertion. The effect of the radon is to cause shrinkage and devascularization, and in two cases the growth has apparently disappeared entirely. (C) Central application of radium. In this method the growth is dealt with from the center of the lumen which is dilated and a more or less rigid tube inserted, to the outer surface of which radium needles are fixed. It has the advantage of definitely dilating the lumen. It has the disadvantage of dealing with the growth from the center and most degenerated part, and one which is farthest from the active and growing edge. In addition, sepsis on the ulcerated surface is encouraged by the advent of radium.

From the endoscopical point of view gastrostomy is an expression of complete failure. Such a failure occurs frequently; patients are never grateful for having had the operation carried out; neither does it render subsequent endoscopical radium treatment easier or more satisfactory. The proper use of diathermy, notwithstanding its failure to cure will in the great majority of cases maintain the patency of the lumen sufficient to maintain fluid nourishment.

The following points called for further investigation. Is it possible by stimulation of a periesophageal infiltration to increase the safety and efficiency of diathermy? What variations in radium technic should be adopted as to point of insertion, dosage and screenage? What types of the esophageal tumors are particularly radiosensitive inasmuch as some tumors appear to be very radiosensitive and others to be entirely unaffected?

Variations in technic for different areas may render the results with treatment of carcinoma of the esophagus analogous to those in carcinoma of the mouth.

Nehrkorn, A.: Zur Entfernung von Fremdkörpern aus dem Oesophagus. (Removal of Foreign Bodies From the Esophagus.) Deutsche Ges. f. Chir. Sitzg. 8: 11, IV, 1931.

A case of foreign body in the esophagus removed through a posterior mediastinotomy approach is reported. Mention is made of Von Endelen who performed the operation five times for such cases. This case was that of a man of twenty-one, who swallowed a razor blade. It became tightly fixed in the esophagus at a point opposite the bifurcation of the trachea. Because of its fixation and immobility, operative interference was indicated. The procedure employed consisted in wide resections of portions to the 3rd to 9th ribs with portions of the transverse vertebral processes. A large inflammatory swelling of the posterior esophageal wall was brought into view in which at first the foreign body could not be located. A stomach tube was then passed and an incision approximately 3 cm. long made down upon it over a suspicious area in the inflammatory swelling. Through this the razor blade was located and extracted. Suture of the defect was very difficult because of the edema and fragility of the inflamed wall of the esophagus. An esophageal fistula was present for a short time postoperatively. In all 7 cases of swallowing of razor blades have been seen in the Elberfelder Clinic.

Anschütz: Recommends an approach through the stomach for foreign bodies which have lodged below the bifurcation near the cardia. He reports three cases so managed.

Eiselberg has had 12 such cases. Diverse articles are swallowed, those usually necessitating removal being metallic and sharp edged. One case swallowed a foreign body on three successive occasions and each time required an operation for its removal.

Trachea, Lungs and Pleura (Nontuberculous)

Ernst, N.: Beitrag zur Behandlung der Brustfell-Lungenverletzungen. (Treatment of Chest and Lung Injuries.) Deutsche Ztschr. f. Chir. 231: 578, 1931.

Although it has been more than ten years since the war nevertheless most of the literature upon wounds and injuries of the chest and lung reflect the treatment as practised during warfare. The methods that have been described are applicable only to war injuries because of the great differences in the type of injury and in the facilities for their treatment from that in civil life. In civil life the projectiles are of smaller caliber and are traveling at less velocity, contamination is less and usually of minor degree, the extent of the wound is not so widespread, and the type of surgery does not have to be of such an emergency and time-limited character.

The article is based on an analysis of 169 recent traumatic lesions of the chest and lungs seen in Sauerbruch's Clinic (Munich) from 1919 to 1927. The various modes of treatment for different types of trauma are briefly discussed. The anatomo-physiologic peculiarities of the chest must always be kept in mind. Shock is almost always a complete contraindication for interference except in clearly indicated cases. If cases are seen twenty-four hours after injury, expectant treatment is usually best. The prime indications for operative treatment are progressive internal hemorrhage and tension pneumothorax. Active measures to evacuate hemothorax are valueless and dangerous. The conclusion is drawn that operative interference in wounds of civil life is far rarer than in war wounds and that by far the best results attend expectant treatment.

Klages, F.: Uber die Chondrome der Lunge. (Chondroma of the Lung.) Bruns' Beiträge 151: 661, 1931.

Chondroma as a benign pulmonary neoplasm has been almost exclusively known as a manifestation of chondromatous neoplasm of contiguous thoracic viscera. Development in pulmonary surgery and diagnostic powers of x-ray have thrown a new light upon the subject and rejuvenated clinical interest in this tumor. The author had the opportunity of making a clinical diagnosis of pulmonary chondroma by x-ray. The plate showed a large spheroidal shadow in the right lung field approximately 7 by 6 cm. in size. The shadow was sharply demarcated from the contiguous normal air containing parenchyma, standing out in sharp contrast. The opacity cast by the neoplasm was of uneven density, through it were scattered irregular, large, very dense, flaky shadows which alternated with clear areas. Upon this picture the diagnosis of pulmonary chondroma was made. At operation (thoracotomy with resection of the fourth and fifth ribs) an apple-sized neoplasm of the lower lobe was found which was visible upon the anterior surface of the lung. The lobe was brought into the operative wound, the neoplasm and the surrounding pulmonary parenchyma were circumcised and excised en bloc. The wound margins were approximated and sutured together and the pleural opening closed air-tight. The defect was filled in with a flap of breast and pectoral muscle. The wound went on to heal perfectly. The histologic diagnosis was myxomatous chondroma with islands of ossification. From a review of the literature of chondroma of the lung it seems that in the past they have usually been confused with echinococcus cyst.

Graberger, G.: Beitrag zur Kenntnis der basalen paramediastinalen Dreieckschatten. (Basilar Paramediastinal Triangular Shadows.) Acta. Rad. XII. 3.15.VII. 67, p. 250, 1931.

The paper is a consideration of the pathogenesis of basal paramediastinal triangular shadows. Lesions and infiltrations of the accessory inferior lobe are an important although almost invariably overlooked explanation for the shadow. Anatomical specimens are described showing the positions and relations of the accessory inferior lobe. Infiltration of this lobe can produce a basal paramediastinal triangular shadow, the sides of which extend in straight lines obliquely upward to the hilus. The differential diagnosis of the basal paramediastinal triangular shadow is discussed, the diagnosis varying between encapsulated exudate, atelectasis, costomediastinal pleuritis and infiltration of the inferior accessory lobe. X-rays of nine cases of the basal paramediastinal triangular shadow are demonstrated. In two, the x-rays show a lateral, well-defined, basal mediastinal infiltration initially, and later a thread-like interlobar line corresponding to the previous lateral margin of the infiltration. Two other cases of bronchiectatic pneumonitis and tuberculous infiltration of the inferior accessory lobe are demonstrated. In five cases only the interlobar line denoting the fissure separating the accessory inferior lobe was present.

Sargent, E., Kourilsky, and Poumeau-Delille: Quelques Reflexions sur le Mode de Guerison des Abces du Poumon. (The Cure of Lung Abscess.) Arch. méd.-chir. de l'app. respir. 5: 312, 1930.

There are three possible methods for the cure of lung abscess:

1. Spontaneous cure, which occurs in some abscesses caused by the streptococcus and pneumococcus through absorption, cavitation and obliteration. This course of spontaneous cure is not that usually pursued by putrid abscesses. The ulcerating necrotic bronchopneumonia, which is the pathologic basis of lung abscess, goes on to the formation of large areas of gangrenous slough which cannot be absorbed. The slough becomes incarcerated in the cavity and acts as an infected sequestrum, maintaining a subchronic or chronic necrotic bronchopneumonia, favoring extension of the process and involvement of the bronchial tree to produce bronchiectasis.

2. Medical cure. Treatment with emetin secures rapid results with amebic abscesses. It has practically no effect in nonamebic abscesses. Spontaneous or medical cure occasionally is seen after a few weeks—very rarely after several months.

3. Surgical cure. The authors recommend surgical intervention for lung abscess, preferably not later than two and a half months after the onset of the disease. This is always undertaken unless definitive spontaneous and appreciable improvement is taking place. Pneumotomy or pneumectomy are the methods of choice. This rule cannot be made too absolute although it holds true in the greater number of cases.

That spontaneous cure may occasionally take place in a putrid abscess is illustrated by a case reported in which a patient with a large putrid abscess of the left lower lobe developed another abscess of the right upper lobe. The abscess of the left lower lobe, being well localized, superficial and subpleural, was drained after 15 days of observation by a simple pneumotomy and went on to cure. The abscess in the right upper lobe went on to spontaneous cure, without interference, inside of three weeks, without any clinical or x-ray sequelae.

Abadie, J.: Dix cas de Kystes Hydatiques du Poumon. (Ten Cases of Pulmonary Hydatid Cyst.) Bull. Soc. Nat. Chir. 57: 124, 1931.

The report is made on a series of ten operated cases of pulmonary hydatid cysts. In the ten cases twelve operations were performed—only one was done in two stages. Four cases were treated with drainage while seven had successful removal of the membranes without drainage. In one case empyema was a complication. Five of the cases were under twenty years of age. The incidence on both sides was equivalent, twice the disease was bilateral, and in one case two cysts were encountered in the left lung. Important aids in the diagnosis are expectoration of portions of membrane and the x-ray findings. The aid rendered by artificial pneumothorax is negligible. The humoral reactions of Weinberg, Fleig and Casoni have no diagnostic value, at most they confirm the diagnosis. Exploratory puncture is to be condemned. In one case syncope followed and in one case death from pulmonary edema.

Treatment. Many cysts, especially those centrally located, cure themselves spontaneously by expectoration of the membrane. Central, noninfected cysts should be left untouched. Central infected cysts, in spite of great risk, should be operated upon. Peripheral cysts, even if uninfected, should be operated upon. Therapeutic puncture, especially with formalin injection, should be discarded. The operative approach in the presence of adhesions is simple. If infection be present the drainage must be ample through wide exposure. If the cyst contents are clean, aseptic—the so-called "dead cyst" without signs of suppuration, the cyst is emptied and a small drain inserted at the lower angle and the wound closed over in layers. If everything goes well the tube is removed after three or four days. In the absence of adhesions a pneumothorax is to be feared because of the danger of empyema and because of the interfering action of a pneumothorax on marsupialization. Usually these cases, however, are done in one stage. The approach is through a rib resection or through an intercostal thoracotomy using a rib spreader. It is advised to suture the closed cyst cavity to the chest wall incision to anchor the lung to avoid postoperative complications. Air in the cyst and pleura is best aspirated. Bronchial fistulas were not observed. The use of differential pressure apparatus was not necessary.

Trachea, Lungs, Pleura (Tuberculous)

Gale, J. W., and Middleton, W. S.: Scaleniotomy in the Surgical Treatment of Pulmonary Tuberculosis. Arch. Surg. 23: 1, July, 1931.

The scalene muscles anchor the first ribs for the action of the upper three intercostal groups. Through their influence the intercostal muscles tend to bring about a bucket-handle movement of these ribs—increasing the lateral and anteroposterior dimensions of the thorax but if unopposed such intercostal action results in shortening of the longitudinal axis of the thorax. With experimental paralysis of the scaleni the most striking features are caudad movements of the upper three ribs and exaggerated outward movement of the costal margins on inspiration. On quiet inspiration the scaleni do not elevate the first ribs but by their tone they neutralize the caudad tendency of intercostal contraction in this location.

The anatomy of the scaleni is briefly described. The surgical approach for section of these muscles is somewhat similar to the one employed in the approach for exposure of the phrenic nerve. Section of each muscle is made near its insertion. Local infiltration anesthesia is used and the cautery knife is employed for hemostatic section.

After phrenicotomy there is often increased intercostal and scalene activity. The chief therapeutic effect of phrenicotomy is realized through diminution of the longitudinal axis of the thorax and relaxation of the affected lung. The performance of simultaneous scaleniotomy and phrenic block is therefore proposed.

Seven cases of pulmonary tuberculosis have been subjected to scaleniotomy. In the first two cases phrenic block was followed by scaleniotomy one week later. In the remaining cases a combined operation was done. In every case there was a marked reduction in the respiratory excursion in the upper portion of the thorax. The caudad movement of the upper three ribs has not yet been seen in any of these patients, but there was an absence of increased upper intercostal movement so commonly seen when phenicotomy alone is performed. Observations on this group of patients has not been over a sufficient period of time to warrant final conclusions.

The authors conclude that the increased activity of the upper part of the thorax after diaphragmatic paralysis indicates the need for scalenicotomy for satisfactory immobilization of the involved apex. The combined operation of scaleniotomy and phrenic block induces apical rest coincident with diaphragmatic paralysis.

Proust and Maurer: Radiographie d'apicolyse. (Radiography of Apicolysis.) Bull. et mém. Soc. nat. chir. 57: 1155, July, 1931.

The authors report on a series of cases of apicolysis without plombage. The use of grafts is unnecessary because the apex of the lung when separated from its attachment to the thoracic cage will be depressed and contracted by the exercise of its own retractile tendency. With depression of the bony thoracic cage—the natural retractile elasticity of the lung can come into play.

Apicolysis, as performed in the authors' technic, consists in a posterior approach with resection of the transverse apophyses juxtavertebrally completed by a resection of the anterior extremities and costal cartilages of the first two ribs through a parasternal incision.

The postoperative x-rays show the obliteration of the apical cavities after such an apicolysis.

The posterior incision extends paravertebrally to the border of the fascial aponeurosis of the trapezius. It is easy to mark out the spinous process of the seventh cervical vertebra which corresponds to the upper portion of the posterior angle of the first rib. An incision is made 7 to 8 cm. long. The trapezius is then divided by careful separation of the fibers. One then comes upon the second transverse apophysis which is laid bare. The costovertebral articulation is then opened—the axis of which is forty-five degrees from the vertical. The apophysis is sectioned flush with the vertebral attachment and it is then easy to excise the entire neck of the rib. To find the first transverse apophysis it is necessary to remember that it lies in a much deeper plane than the second. The first transverse apophysis must be laid bare with extreme care because of the important anatomic relations and fibrous attachments. By careful exposure first of the inferior face of the rib and then carefully of the superior aspect of it—resection of its posterior extremity as far as the tubercle of Lisfranc can be secured. A parasternal approach is then made to resect the remainder of the first and second ribs with the accompanying cartilages as the indications present themselves.

Accompanying x-rays show that this type of apicolysis without plombage or graft suffices to obliterate cavities and effectively depress the apex.

Sachs, W., and Sperl, W.: Ueber die Plombierung bei Lungentuberkulose. (Plombage in Pulmonary Tuberculosis.) Beitr. klin. Tuberk. **74:** 168, 1930.

The basis of the paper is a report of eight cases of plombage for pulmonary tuberculosis. Decisive trustworthy statistics on end-results cannot be given because of the short period of observation (approximately one year). At present four of the patients are able to work and are free of expectoration, the four others are not ready to work although two of these could work part time. Complete riddance of expectoration was obtained in four cases, freedom from tubercle bacilli in three. The complete disappearance of cavitation was noted in only one case in whom the plombe was extruded after nine months. Two cases will come to thoracoplasty because of the development of cavitation where the undersurface of the plombe has melted away. One case showed a ballooning up of the scar caused by the partial extrusion of the plombe which was held in place by massage and a pressure bandage. All the operations were performed by the Ziegler's technic without rib resection. At least three weeks of absolute bed rest are necessary postoperatively to insure healing and fixation of the plombe and to watch for local complications. Two cases developed exacerbations on the opposite side.

The indications for plombage are:

1. As an independent procedure it can be used in bilateral lesions when pneumothorax is ineffective or impossible and one side has a large rigid cavity resisting closure.

2. In cases of upper lobe cavities when thoracoplasty has been contraindicated.

3. When patients refuse thoracoplasty. In these patients plombage may act as a preparatory operation to a later necessary thoracoplasty.

The author's summary is that, at best, plombage, as an independent procedure can only be a substitute when other operative procedures for various reasons are impossible.

Boit, H., and Scholz, H.: Die Behandlung der Lungenkavernen. (Treatment of Lung Cavities.) Ztschr. f. Tuberk. **61:** 193, 1931.

Pulmonary tuberculosis being a systemic disease, it is not held forth that cure of the pulmonary cavity as a local measure will cure the disease. However, the poor prognosis of pulmonary tuberculosis with patent cavitation has often been brightened with collapsing measures.

Because of the great uncertainty of the spontaneous healing of tuberculous cavities and the usual progress of the disease, collapsotherapy should be instituted at the earliest possible moment.

In the presence of free pleural spaces artificial pneumothorax has given excellent results and in some cases is the method of choice.

With faulty pneumothorax or with the impossibility of its introduction, thoracoplasty, the dangers of which have been highly overestimated, should be performed, however, only with careful observance of the well-known clinical indications.

A preliminary phrenicectomy often will permit one to diminish the number of ribs to be resected.

As an independent procedure phrenicectomy has been disappointing because of its failure to have a permanent effect.

As a complementary operation for the completion of the effect of an unsatisfactory paravertebral thoracoplasty, extrapleural pneumolysis and plombage is indicated. This is usually performed through wide resections of the upper anterior ribs with filling of the entire space with paraffin plombe, care being taken not to exert excess pressure. Plombage is initially preferable to thoracoplasty in cases of large upper lobe cavities with widespread adhesions to the pleura with but little involvement of the rest of the lung.

Hoseman, L.: Zur offenen Strangdurchtrennung bei Lungentuberkulose. (The Open Method of Severance of Adhesions in Pulmonary Tuberculosis.) 55. Tag. d. Deutsche Ges. f. Chir. Sitzg. **8**: 11, 1931.

The formation of adhesions is a very great hindrance in pneumothorax treatment. It is extremely hazardous to attempt to break or stretch these adhesions by the use of high tension pneumothorax. The closed method of Jacobeus has many great disadvantages. The improvement in technic lies in exact orientation with the operating field, always under direct vision, obviating the well-known hazards of the closed method. Severance of adhesions should not be done prematurely—at least three or five months should elapse following the first pneumothorax. The operative technic is simple. The thorax is opened with an intercostal incision, exact localization of the adhesions being obtained with stereo x-rays. The adhesions are visualized by use of an elongated lighted rod. The severance of adhesions is performed with a hooked electrode. Diathermy must be employed to avoid bleeding. Bleeding must be avoided because of the frequent occurrence of empyema postoperatively in cases in which there has been considerable oozing. This is an argument of capital importance in favor of open operation in which hemostasis is relatively easy.

Heymer, A.: Zur Thorakokaustik. (Thoracic Cauterization.) Ztschr. f. Tuberk. **59**: 37, 1930.

The article is based on a series of two hundred cases of cauterization of adhesions which have been performed in the past two years at the Beelitz Sanatorium according to Kramer's technic. Stereoscopic x-ray plates were found necessary for the determination of the type of adhesions and for their localization for surgical approach. The types of adhesions found were: 25.4 per cent cord-like, 66.7 per cent membranous, 7.9 per cent broad, sheet-like adhesions. By cauterization of the adhesions a good collapse was obtained in 51.9 per cent, a partial collapse in 37.7 per cent, and no success in 10.4 per cent. Clinically 9.4 per cent were considerably improved, 74 per cent were moderately improved, 16.6 per cent were no better or were worse. Before the procedure 91 per cent had tubercle bacilli in the sputum; after operation only 32 per cent had tubercle bacilli present. In 88 per cent there appeared a trifling exudate in the pleural sinus with a temperature rise of one to two degrees with defervescence in three or four days and resorption of the exudate in ten to twelve days. Such a course was considered the normal postoperative reaction. In 7 per cent there appeared either a moderate or a large effusion which in two cases went on to empyema. Dangerous endothoracic hemorrhage was not observed in these two hundred cases.

Troll, A.: Einige Versuche von Behandlung mit Oleothorax. (Experiences With Oleothorax.) Acta. Med. Scand. **75**: 3-4, 1931.

The influence of oleothorax upon acute tuberculous empyema with secondary infection is not beneficial. These cases are usually complicated by a pulmonary fistula and more often than not have a marked clinical aggravation of symptoms if oleothorax is induced. The best results have been secured in the cases of chronic empyema complicating pneumothorax. There is a marked thinning and clearing of the purulent exudate which action is ascribed not to the antiseptic action of the oil but to the rôle it plays in diminishing the pleural secretion and in producing marked thickening of the pleural layers, particularly the visceral. If, in the presence of an extensive pneumothorax, large apical cavities persist because of adhesions, it is advised that these adhesions be cauterized to permit collapse of

the cavities before oleothorax is induced. If cavities are present at the base phrenicectomy is performed prior to the oleothorax. Injections of 2000 to 2400 c.c. are well borne. Large doses are dangerous and should not be given.

Pissavy, A.: Traitement des pleuresies purulentes tuberculeuses malignes. (Treatment of Malignant Purulent Tuberculous Pleurisy). Presse méd. **18**: 1060, 1930.

The author presents an analysis of some compiled statistics on the treatment of malignant purulent tuberculous effusions. He reports on four methods as follows:

I. Aspiration with irrigation (methylene blue, iodine, oil, crisalbin), 15 cases, all with pneumothorax. In 9 of these cases the pleuritis was on the basis of the spontaneous perforation occurring with spontaneous pneumothorax. Of these 15 cases, 11 died (mortality 73 per cent) and two had closure of their fistulas.

II. Oleothorax. The statistical analysis does not prove it to be of as great value as has been thought. Twenty-one cases; of which 13 died (mortality 62 per cent), 4 improved with closure of fistula, 1 improved with patent fistula, 2 were made worse.

III. Pleurotomy. Seventeen cases of whom 11 died (mortality 64 per cent), 2 cases cured, 1 improved, 1 slightly improved.

IV. Thoracoplasty. The mortality of a large combined series of thoracoplasty for purulent malignant pleuritis is 60 per cent.

In conclusion the author states that a distinction between benign and malignant pleurisy is not always justified.

The results of the different types of treatment appeared nearly equivalent; it is difficult to decide which one is to be preferred.

Kirschner, A.: Die Ligatur der Vena Pulmonis bei Lungentuberkulose. (Ligation of the Pulmonary Vein in Tuberculosis.) Deutsche ges. f. Chir. Sitzg. v. 8-11, IV, 1931.

Ligation of the pulmonary vein was suggested to the author in consideration of the well-known clinical fact that patients with cardiac disease in whom there is stasis in the lesser circulation almost never contract pulmonary tuberculosis. Tiegel has pointed out in a number of animal experiments that it is very probable that through stenosis of the pulmonary veins to a lobe, the resistance to tuberculosis of the lobe concerned can be materially heightened. Up until now four patients with cavernous cirrhotic lesions of the upper lobe have been treated by this procedure, the technic of which is briefly described. The operation was without complication and the patients have shown appreciable improvement in their lung findings. The follow-up of the course of these cases has been short but is highly encouraging.

Horing, F. O.: Ueber den Wert der Phrenicusexäirese als selbständigen Eingriff bei der Lungentuberkulose. (The Value of Phrenicectomy as an Independent Procedure in Pulmonary Tuberculosis.) Beitr. klin. d. Tuberk. **78**: 115, 1931.

The author reports 56 cases of phrenicectomy as an independent procedure for pulmonary tuberculosis. It is pointed out that very strict criteria must be observed to clearly evaluate the worth of a procedure such as phrenicectomy in pulmonary tuberculosis.

In cavernous tuberculosis pneumothorax should always precede phrenicectomy with the exception of middle or lower lobe cavities.

If, three or four months after a phrenicectomy, the cavity has not been appreciably influenced—other methods of collapsotherapy should be employed, preferably a thoracoplasty.

In the treatment of pulmonary tuberculosis, phrenicectomy as an independent procedure, must be considered as an experimental attempt which only rarely can meet with success, not as a method in tuberculosis therapy with which one can content oneself. One should not be detoured by experimentation with phrenicectomy in the proper treatment of pulmonary tuberculosis. However, phrenicectomy has this virtue, that it is an active measure of treatment and through it further collapsotherapy is brought to mind. It should always be realized that the outcome of collapsotherapy should not be improvement but cure, and that the necessity of returning the patient back to society should always be in the foreground. Improvement through phrenicectomy should not be substituted for the cure which might come about through more active surgical collapsotherapy.

In 56 cases the often claimed, favorable action upon upper lobe cavities was frequently not demonstrated. Recent thin-walled cavities are much more amenable to collapse by phrenicectomy than are old, rigid, fibrotic cavities. An extreme high position of the diaphragm which usually can be expected if the pleural space is free, is not always an insurance against a later exacerbation of the lesion. In conclusion the author states that strict statistical studies should be made of the effects of phrenicectomy as an independent procedure in collapsotherapy and the indications for its use as a sole procedure be limited to few selected cases.

Oeri, F.: Phrenicusexäirese. (Phrenicectomy.) Schweiz. med. Wchnschr. p. 131, 1931.

The author summarizes the indications and contraindications for phrenicectomy as follows:

1. Contraindications:

(1) Undrained empyemata.

(2) Intestinal tuberculosis.

(3) It is very doubtful whether the operation is of any effect upon large, rigid walled upper lobe cavities.

2. Indications:

(1) The method of choice in clear-cut lower lobe lesions.

(2) As the substitute and necessary expedient in failure of pneumothorax when a free pleural space is no longer present. In consideration of the comparison between phrenicectomy and pneumothorax, phrenicectomy has the advantage of an immediate encroachment but its success is not predictable nor its effect to be relied upon. The operation should not take the place of pneumothorax. If pneumothorax is impossible because of the absence of a free pleural space, phrenicectomy should be employed before a plastic is contemplated.

(3) As a preliminary operation when a thoracoplasty is decided upon as the necessary procedure.

(4) As a supplementary operation for incomplete pneumothorax or unsatisfactory thoracoplasty.

(5) To produce a milder degree of collapse particularly where the lesions are bilateral.

(6) It is of value at the end of pneumothorax to maintain the degree of collapse secured and to prevent the expansion of the lung in filling the vacated pleural space.

(7) As a preliminary trial operation before the performance of thoracoplasty.

(8) For severe pulmonary hemorrhage.

The American Association for Thoracic Surgery

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The Fourteenth Annual Meeting of the American Association for Thoracic Surgery

THE fourteenth annual meeting of the American Association for Thoracic Surgery was held in San Francisco July 1, 2 and 3, 1931. Perhaps the most important official transaction at this meeting was the establishment of an official organ, The Journal of Thoracic Surgery. The scientific program was of the usual high order and will be published in this journal.

The "extra-scientific" program given by Doctors Brunn, Eloesser, and Holman was delightful. The entertainment, both for the members and for their wives, was sufficiently developed to merit the printing of a special program. The dinner given at the home of Dr. and Mrs. Brunn for the members and their wives was of such proportions as to require a battery of noted Chinese chefs and a group of entertainers, necessitating removal of all their furniture including the piano (the entertainers insisted on bringing their own). The special yacht which was at our disposal for the trip to Paradise Cove and the Barbecue on the last day of the meeting were performed according to the technic of Eloesser.

The following officers were elected for the year 1932:

Dr. Frederick T. Lord, Boston, President
Dr. George P. Muller, Philadelphia, Vice President
Dr. Duff S. Allen, St. Louis, Secretary
Dr. Carl Eggers, New York, Treasurer

Dr. Emile Holman, San Francisco, was elected as the new member of the Council to fill the vacancy of Dr. David A. Stewart, Ninette, Manitoba, who automatically retires January 1, 1932, having served four years.

The following active members retired to the rank of senior membership:

Dr. Lawrason Brown, Saranac Lake
Dr. Rufus Cole, New York
Dr. Victor P. Diederich, Hot Springs
Dr. H. Kennon Dunham, Cincinnati
Dr. Edmond M. Eberts, Montreal
Dr. Max Einhorn, New York
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Dr. Eugene H. Poole, New York
Dr. John Smyth, New Orleans
Dr. Franz Torek, New York
Dr. J. H. Wilms, Cincinnati

The following were elected to active membership:

Dr. Frank B. Berry, New York
Dr. Claude S. Beck, Cleveland
Dr. Dan C. Elkin, Atlanta
Dr. Deryl Hart, Durham
Dr. Edward J. O'Brien, Detroit
Dr. C. M. Van Allen, Pieping, China

The following were elected to associate membership:

Dr. Alfred Blalock, Nashville
Dr. Selling Brill, San Francisco
Dr. B. Noland Carter, Cincinnati
Dr. Richmond L. Moore, New York
Dr. Richard Overholt, Boston
Dr. Owen H. Wangensteen, Minneapolis